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THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

APRIL, 1925

COMMUNICATIONS

TONOMETRY*

BY

PROFESSOR HJ. SCHIÖTZ

OSLO, NORWAY

Fresh Corrected Graphs

IN my previous treatise on tonometry in THE BRITISH JOURNAL OF OPHTHALMOLOGY for May and June, 1920, I gave an account of measurements made on ten dead eyes *in situ*. These measurements coincided fairly well with those made by Mr. Priestley Smith with the 5.5 gm. weight on twelve dead eyes *in situ*, published in the *Ophthalmic Review*, March, 1915. A graph drawn up according to these last-named measurements stood about 4 mm. Hg. higher than the graph hitherto applying to that weight which was based on measurements of eight enucleated eyes.

Priestley Smith's measurements, and later my own, proved that the graphs hitherto used stand too low and ought to be amended; my opinion, however, was that a considerably larger number of measurements should be made prior to constructing and publishing fresh graphs for clinical use. This I have now done. During the last year I have measured thirty dead eyes *in situ*, and on this basis I have compiled the fresh graphs now published.

For these measurements I used my old standard tonometer, which had been used for all my previous measurements, and which is always used as the standard for the adjustment of other tonometers.

*This article is an abridgment, by Professor Schiötz, of a more detailed paper which was published in *Acta Ophthalmologica*, Vol. II, fasc. I. Table III is now added.

In all the later measurements the canula was carried into the anterior chamber, as indicated in the above-named treatise (p. 255, 1920), being inserted a couple of millimetres outward from the edge of the cornea and pushed forward through the iris and into the region of the pupil without injuring the lens, the opening of the canula being forward. The outside diameter of the hollow needle was 1 mm., that of its lumen 0.5 mm. On raising and lowering the manometer, the fluid (normal saline) flows with ease in and out of the anterior chamber, and not once was there any stoppage of the canula or irregularity in the pressure-variations.

Of the thirty eyes, twenty were in individuals who had been dead from 8 to 48 hours prior to the measuring; on the remaining ten the measurements were made one to two hours after death. Several measurements were taken for each pressure-height. In the following table are entered the maximum, minimum, and average values for all the pressures, and the two groups mentioned above are separated.

TABLE I

THE OPEN EYE—*i.e.* TAP BETWEEN EYE AND MANOMETER OPEN

Pressure H_2O.	Group of 20 eyes measured 8 to 48 hours after death. Deflection of Pointer.			Group of 10 eyes measured 1 to 2 hours after death. Deflection of pointer.		
	Maximum	Minimum	Average	Average	Maximum	Minimum
			5.5 gm. weight			
20 cm.	23.5	15.3	**18.98**	**19.2**	22	17
30 ,,	13.5	7.47	**10.27**	**10.93**	12.3	9.5
40 ,,	7.2	3.87	**5.79**	**6.25**	7	5.33
50 ,,	4.2	2	**3.27**	**3.57**	4.48	2.5
60 ,,	2.3	0.6	**1.55**	**1.63**	2.2	1
70 ,,	1	0.1	**0.37**	**0.2**	0.6	0
			7.5 gm. weight			
30 ,,	19.5	13	**15.5**	**15.1**	18	13.75
50 ,,	7.75	5.3	**6.53**	**6.77**	8	5.5
70 ,,	3.8	2	**2.85**	**2.8**	3.5	2
90 ,,	1.5	0	**0.7**	**0.7**	1.75	0.2
100 ,,	0.7	0	**0.3**	**0.33**	—	—
			10 gm. weight			
50 ,,	13	9	**10.5**	**10.28**	11.7	9.5
70 ,,	6.7	5	**5.9**	**5.48**	7	5
90 ,,	3.8	2	**3.12**	**3.06**	4.5	2.5
110 ,,	1.5	0.5	**1.1**	**1.2**	2	0.9
120 ,,	—	—	**0.3**	**0.44**	0.5	0.1
			15 gm. weight			
50 ,,	20	16	**18.45**	**17.39**	20	15
70 ,,	14	10	**11.74**	**10.8**	14	9
90 ,,	8.5	6.5	**7.65**	**7.08**	7.9	6.2
110 ,,	6	3.5	**5.2**	**4.74**	5.8	3.8
130 ,,	4	2.1	**3.47**	**3**	3.75	2.2
146 ,,	—	—	**2.7**	**2.2**	—	—

THE CLOSED EYE—*i.e.*, TAP BETWEEN EYE AND MANOMETER CLOSED

			5.5 gm. weight.			
20 Cm.	12.6	5.6	**8.41**	**7.67**	9.47	6.2
30 ,,	7	3.7	**5**	**4.79**	5.75	4.1
40 ,,	4.1	1.86	**2.93**	**3.1**	4.25	2.4
50 ,,	2.6	0.85	**1.56**	**1.8**	2.97	1.1
60 ,,	1.2	0.10	**0.43**	**0.55**	1	0.17
70 ,,	—	—	**0**	**0**	—	—
			7.5 gm. weight.			
30 ,,	9.5	5.5	**7.3**	**7.1**	8.67	6.55
50 ,,	5	2.7	**3.82**	**3.8**	4.5	3
70 ,,	2.5	0.87	**1.63**	**1.55**	2.3	1
90 ,,	1	0	**0.32**	**0.22**	1.1	0.2
100 ,,	—	—	**0**	**0**	—	—
			10 gm. weight.			
50 ,,	7	4.87	**5.86**	**5.47**	6.4	4.2
70 ,,	4.6	3	**3.74**	**3.43**	4.6	2.9
90 ,,	2.9	1	**2.12**	**1.97**	3	1.5
110 ,,	—	—	**0.3**	**0.55**	0.9	0.2
120 ,,	—	—	**0**	**0**	—	—
			15 gm. weight.			
50 ,,	11.3	8.23	**9.96**	**9.3**	10	8.3
70 ,,	8.5	5.9	**6.85**	**7.1**	8	6.1
90 ,,	5.83	4	**5.16**	**4.53**	6.1	4.1
110 ,,	4.4	2.5	**3.63**	**3.38**	4	3
130 ,,	3.15	1.5	**2.56**	**2.21**	3	1.6
146 ,,	—	—	**2.1**	—	—	—

TABLE II

AVERAGES OF THE TWO GROUPS COMBINED.

Pressure H_2O	5.5 gm. weight. Deflection of the Pointer. Open	5.5 gm. weight. Deflection of the Pointer. Closed	7.5 gm. weight. Deflection of the Pointer. Open	7.5 gm. weight. Deflection of the Pointer. Closed	10 gm. weight. Deflection of the Pointer. Open	10 gm. weight. Deflection of the Pointer. Closed	15 gm. weight. Deflection of the Pointer. Open	15 gm. weight. Deflection of the Pointer. Closed
20 cm.	19.09	8.4						
	(19.58)	(8)						
30 ,,	10.6	4.89	15.3	7.2				
	(10.51)	(4.9)	(15.73)	(7.2)				
40 ,,	6.02	3.01						
		(3)						
50 ,,	3.42	1.68	6.65	3.81	10.39	5.66	17.92	9.63
	(3.26)	(1.67)		(3.8)	(10.59)	(5.9)	(18.8)	(8.47)
60 ,,	1.59	0.49						
	(1.45)	(0.5)						
70 ,,	0.28	0	2.82	1.59	5.68	3.58	11.27	6.97
	(0.13)		(2.74)	(1.6)		(3.6)	(11.26)	(6.3)
90 ,,			0.7	0.27	3.09	2.04	7.36	4.84
			(0.6)	(0.1)	(2.94)	(1.96)		(4.73)
100 ,,			0.31	0				
110 ,,					1.15	0.42	4.97	3.5
					(1.2)	(0.71)	(4.87)	(3.5)
120 ,,					0.37	0		
					(0.54)			
130 ,,							3.23	2.38
							(3.15)	(2.38)
140 ,,							(2.45)	(1.9)
							2.45	2.1 ?
145 ,,							(2.17)	(1.65)

It will be noticed that there is no important difference between the two groups as regards the deflections, I have therefore put

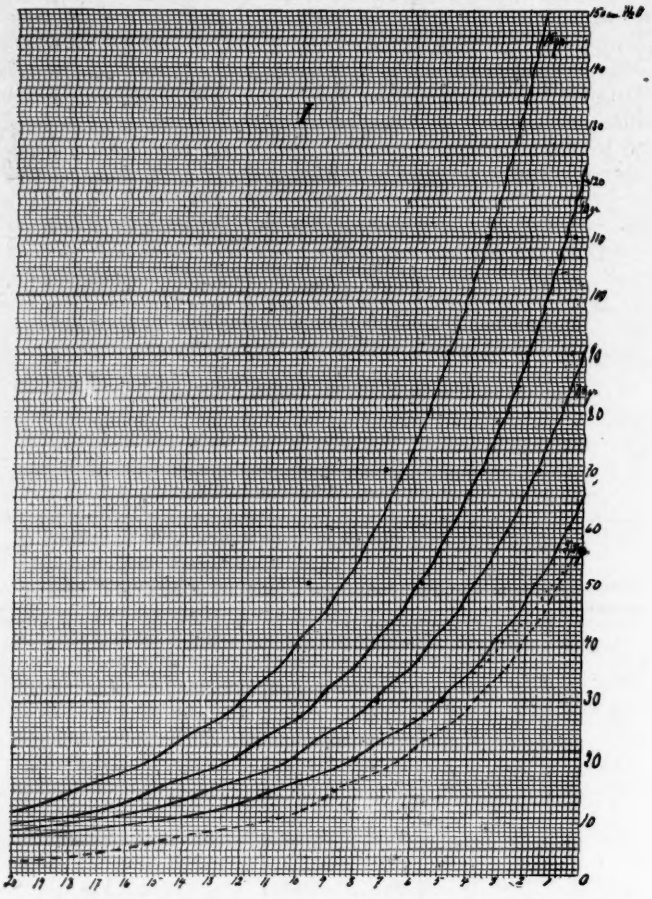
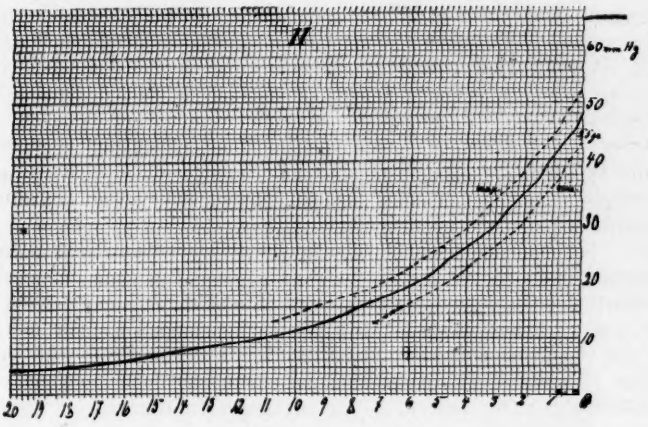


FIG. 1

them together, and in Table II, I have combined their averages. These values can be checked partly by means of special graphs and partly by calculation. By these means I have been able to correct somewhat the figures obtained by measurement; the corrected values are put in brackets below the measured values.

The four curves for the closed eye, given in Fig. 1, are constructed on the basis of these corrected values. The small crosses indicate the measured values entered in Table 2, and as will be seen they mostly appear on or near to the graph line; only the two bottom values for the 15 gm. graph deviate somewhat more than desirable. Notice that these are water-pressure graphs. Below the 5.5 gm. line is a broken line drawn according to the old measurements. These lines run quite parallel. There is also a dotted line which crosses the new graph line between the deflections 4.5 and 5; lower down it lies a little over the new



Deflections of the pointer. Mercury Pressures

FIG. 2

graph, whereas above it slants across to the 0 point of the old graph. This line is drawn according to Priestley Smith's measurements. The discrepancy is undoubtedly owing to the tonometer which he used not measuring exactly in accordance with my standard tonometer.

When graphs are drawn for the "open eye" and compared with the old ones, one notes that at the upper ends (deflection 0) they nearly coincide, but in their downward course they diverge, the fresh ones passing somewhat below the old ones. This struck me very much, but as the divergence is the same in all the four graphs, the cause must be one and the same; undoubtedly it is owing to the fact that for the last measurements I made use of a comparatively fine hollow needle, whereas for the old ones I used

a canula of very large bore. This would influence the curves for the "open eye," but of course not those for the "closed." In Fig. 2 the continuous line is the new graph for the 5.5 gm. weight. The broken lines above and below it are the maximum and minimum graphs for the same weight; they show the range of deflection which may occur with each degree of pressure; they were obtained by averaging the majority of the largest deflections, and of the smallest deflections, in the whole series of thirty eyes, and by correcting the curves in accordance with the main curve.

We may safely assume that for each degree of pressure the maximum and minimum deflections in living eyes will be about the same as we have found on dead eyes. Notice that the interval between the maximum and minimum graphs is not small; thus, with 29 mm. Hg. one may have, in different eyes, deflections varying from 2.2 mm. to 4 mm.; with 15 mm. Hg. one may have deflections from 6.2 mm. right up to 9.4 mm. Consequently with a deflection of 3 mm. (5.5 gm. weight) the pressure will as a rule no doubt be about 29 mm. Hg., but that deflection might be properly referable to the minimum graph and to an intraocular pressure of 26 mm. Hg., or to the maximum graph and a pressure of 33 mm. Hg. This should absolutely be borne in mind when using a tonometer; the deflection of the pointer does not indicate a precise pressure but a pressure-range, which for the 5.5. gm. weight is about 6 to 8 mm. Hg., for the 7.5 gm. weight 8 to 10 mm. Hg., for the 10 gm. weight about 13 mm. Hg., and for the 15 gm. weight about 15 mm. Hg., nor can it be otherwise when we take into consideration the individual peculiarities in the size of the eyes and in the thickness and elasticity of their walls.

In Fig. 3 are the four new graphs for the respective weights of 5.5 gm., 7.5 gm., 10 gm., and 15 gm., the ordinates indicating the height of the pressure in mm. Hg. and the abscissae the deflection of the pointer.

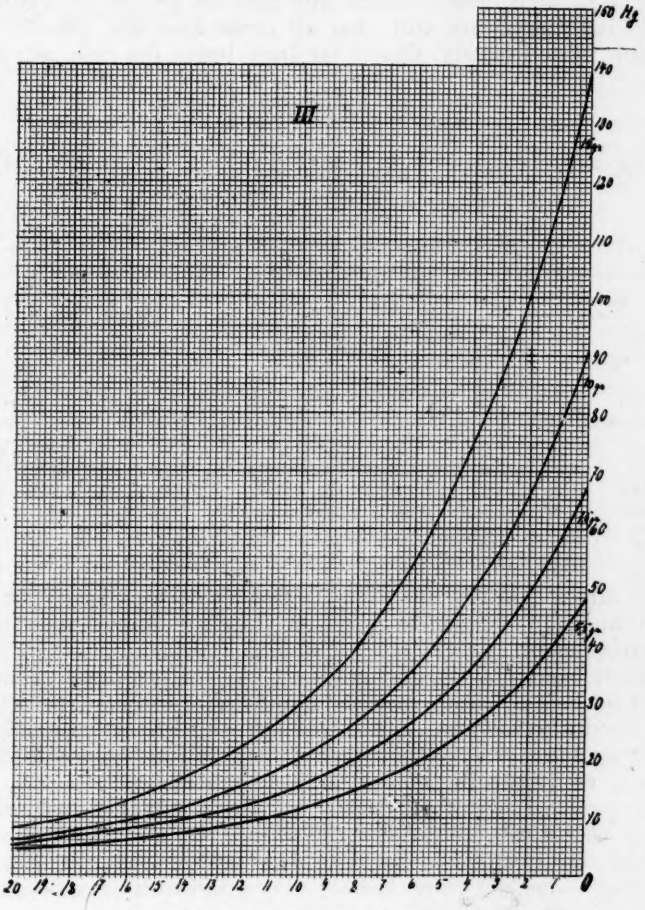
These graphs of 1924 are the final ones for my tonometer with a concave surface at the lower end of the plunger.

In my previous treatise I drew attention to the fact that the position of the graphs does not make any difference in the use of the tonometer. A deflection of 3 mm. will now as previously be the approximate limit for the normal intraocular pressure, but its mercury value is about 4 mm. Hg. higher than previously assumed. For anyone who has recorded the deflections as the measure of the intraocular pressure, which is really the only correct method, the change in the graphs makes no difference, whereas those who have translated the deflection values into mm. Hg. must now rectify these according to the fresh graphs.

Improvement of the Instrument

I have made several attempts at improving the tonometer with the result that I have in principle retained the old model, whereas the appearance has been somewhat altered as is proved by the adjoining drawing. A small simplification, which may be considered an improvement is, that only the 5.5 gm. weight is

Fresh corrected graphs for the Schiötz Tonometer, 1924



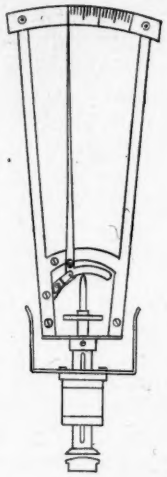
Deflections of the pointer. Mercury Pressures

FIG. 3

now made to screw on and off; the other weights are placed directly on to the 5.5 gm. weight, so that the troublesome screwing on and off of fresh weights for higher pressures is avoided. Further, the sliding cylinder with the small round discs is replaced by a cylinder with ball-bearings; this makes the cylinder more solid, and the tonometer is better balanced.

Accurate Adjustment

It is essential, however, not only that the graphs be accurate, but, more important still, that all tonometers act alike. At present, unfortunately, this is far from being the case, nor will



it be attained unless one or more centres be established for the adjustment of tonometers. In case there are several there must be co-operation between them so that all adjusted tonometers shall measure exactly alike, that is to say like the standard tonometer used by myself, with which the graphs were made. The firm of A. Tandberg has now, in co-operation with myself[1], established such a centre to which tonometers may be sent for adjustment; it also negotiates the sale of adjusted tonometers[2].

In the *Klinische Monatsblätter für Augenheilkunde*, volume 71, page 603 (1923), Messrs. Arnold and Karpow in Tübingen published a treatise "Ueber Aichungsverfahren (gauging process) für das Tonometer von Schiötz." They find such large deviations

1. Professor Hj. Schiötz, Dr. Med. I. Schiötz and Diplom Engineer Tandberg.
2. Applications to be made to Engineer Arnt Tandberg, Akersgaten, Oslo, Norway.

in the deflections of the various tonometers that they recommend simply an adjustment of all instruments to the 3 mm. deflection of the standard tonometer, that is to the assumed limit for the normal intraocular pressure; for when this has been done all smaller deflections will be pathological and will indicate too high an intraocular pressure. This is quite a practical suggestion, but it will not give a really ideal adjustment. Most of the tonometers of various manufacture can be made correct by minor alterations, but to this end exact comparison with a reliable standard tonometer is essential.

TABLE III

THE SCHIÖTZ TONOMETER

DEFLECTIONS OF POINTER AND INTRAOCULAR PRESSURES CORRESPONDING APPROXIMATELY THEREWITH

N.B.—Mercury pressures are in heavy figures

	Weight 5.5 gm.		Weight 7.5 gm.		Weight 10 gm.		Weight 15 gm.	
	Pressure		Pressure		Pressure		Pressure	
Deflections.	cm. H_2O.	mm. Hg.	cm. H_2O.	mm. Hg.	cm. H_2O.	mm. Hg.	cm. H_2O.	mm. Hg.
0	65	**48**	91	**67**	123	**91**	188	**139**
0.5	60	**44**	84	**62**	114	**84**	174	**128**
1	55	**41**	77	**57**	105	**77**	160	**118**
2	47	**35**	66	**49**	90	**66**	137	**101**
3	40	**30**	56	**41**	76	**56**	117	**86**
4	34	**25**	48	**36**	66	**49**	101	**75**
5	29	**22**	41	**31**	56	**41**	86	**64**
6	26	**19**	36	**26**	48	**36**	73	**53**
7	23	**17**	31	**23**	41	**31**	62	**46**
8	20	**15**	27	**20**	36	**26**	53	**39**
9	18	**13**	24	**17**	31	**23**	46	**34**
10	15	**11**	20	**15**	27	**20**	40	**29**
11	13	**10**	18	**13**	23	**17**	34	**25**
14	10	**7**	13	**9**	16	**12**	23	**17**
20	6	**4**	7	**5**	8	**6**	11	**8**

ON THE DEVELOPMENT OF THE EXTRA-OCULAR MUSCLES IN SOME MAMMALS

BY

PROFESSOR OTOKAR LEŠER, M.D.

CZECHOSLOVAK OPHTHALMOLOGICAL CLINIC, PRAGUE

FROM THE CZECHOSLOVAK INSTITUTE FOR NORMAL ANATOMY

DIRECTOR, PROF. JANOŠIK

THE results of my investigations into the development of the bulbar musculature in man (published in my earlier work) induced me to extend my study also to a number of mammals in respect of which the rich collections of the Czechoslovak Institute for Normal Anatomy in Prague were at my disposal. I was able to make use of a complete series exhausting all the developmental stages of the *Spermophilus citillus* and *Sus domesticus*; in addition to which I always compared the results of my studies with observations made on the *Vespertilio murinus, Canis,* and *Cuniculus.*

As generally known, in lower vertebrates the orbital musculature is derived from the walls of the so-called "head cavities" (Corning,[1], Rex[6], Matys[5], etc.); in mammals, however, it is impossible positively to find such structures as might be homologized with the head cavities of the reptiles, birds, etc. In respect of mammals the development of these particular muscles has been studied only on the embryos of the *Sus domesticus* (Kheil[3], Reuter, Keibel); the stages, however, described by these authors are far too advanced.

For easier understanding I first give a brief account of the muscular development in an embryo 7.6 mm. long, in which the rudiments of the musculature innervated by the n. oculomotorius and n. abducens are already defined to some extent. If in this embryo we trace the n. abducens at its issue from the hindbrain, we shall find that it issues as a number (6—7) of nerve fibres which approach one another, produce a delicate, still undefined bundle extending in a distal and rostral direction towards the spot in which the infundibulum forms on the midbrain, and then pass on to the lateral area in the proximity of the carotid artery. Opposite the infundibulum the hypophysis evaginates out of the pharynx. In the indicated spot, near the carotid, the nerve fibres become lost in a condensation of mesoblastic cells. This condensation is sufficiently well-defined and passes as a spindle-shaped structure to the rostrum and to the lateral area towards where the peduncle of the optic vesicle is situated. The process for the development of the upper jaw is still very slight,

in consequence of which this part of the first muscular development seems to be situated in close proximity to the palate. At this stage the n. oculomotorius is much more vigorous than the n. abducens, extends ventrally, closely adjoins the jugular vein, and lodges upon the aforementioned cellular condensation deposited around the n. abducens. On section the cellular condensation forms an oval group of cells, rather large and distinctly circumscribed, in which the laterally deposited group obviously pertains to the n. abducens, the medial group pertaining to the n. oculomotorius. This fusiform structure, equally dependent on the two nerves, continues its course towards the peduncle of the optic vesicle and nearly to the vesicle itself. The n. oculomotorius enters the cellular group much nearer the eye than does the n. abducens. The cellular group treated of

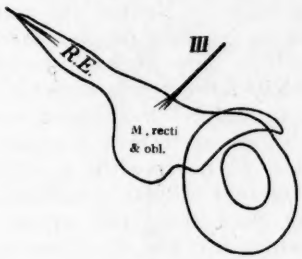


FIG. 1.

Embryo 7.6 mm. of the Spermophilus citillus

above extends in two to three offshoots into the other mesoblastic cells near the eye, and, in consequence, its termination is not distinct.

After the discovery of this obvious commencement of the development of the ocular musculature, it was necessary to look for earlier stages. I set out to examine the stages of two or three mesoblastic somites in order that I might ascertain the origin of the cells producing musculature. It can be safely declared that in embyros of 1-3 mm. it is not at all possible to observe any such structures as might produce the muscle plate beyond the trigeminal ganglion by the otic vesicle and the adjacent ganglions—in the place of future generation of musculature. Only after the length of about 4 mm. has been attained a somewhat denser group of mesoblastic cells can be discovered in the indicated locality, just in the angle inclosed by the carotid and jugular, as high up as where the notochord terminates in the pharyngeal portion of the embryo.

Let us examine an embryo 3.7 mm. long, just at the stage of invagination of the primary optic vesicle; the lens appears as a thickening of the epiblast tending towards the vesicle. At this developmental stage the n. oculomotorius is founded on a number of fibrils issuing from the brain and closely adjoins the jugular vein in its continued course. However, it does not yet reach the group of rather condensed cells between the carotid artery and the vein. It is not yet possible to trace the n. abducens which is visible only at the very beginning of its issue from the brain. The rather condensed cells mentioned above, which are not distinctly differentiated from the surrounding mesoblastic cells, are deposited just where the first evagination for the hypophysis, and the evagination on the brain for the infundibulum are situated.

An embryo 4 mm. long shows no particular changes as compared with the preceding stage. Neither the oculomotorius nor the abducens extend as far as their proper place. However, in the next stage—only a little older (4.5 mm.)—when the secondary optic vesicle is already formed (the anterior wall of the primary vesicle being already deeply invaginated towards the posterior wall) and the lens beginning to form a vesicle which is still open wide in the epiblast, we observe the n. abducens at this stage passing into the aforesaid cellular condensation. The n. oculomotorius extending close along the jugular vein does not yet reach the cells which are not yet distinctly defined in their surroundings, and towards the eye becomes utterly lost amongst the other mesoblastic cells.

At the next stage of development, in an embryo of 5.5 mm. the changes we find are more striking. The n. oculomotorius is much more vigorous, forming a solid nerve bundle, the n. abducens is by far the thinner. In section the plate of condensed cells is larger and forms a definite island of concentrically arranged cells, which extends along the carotid and is surrounded by a dense network of veins on the lateral side. According to the arrangement of cells at this stage it is already possible to observe that the cells deposited on the exterior for the most part belong to the n. abducens, while those situated medially belong to the n. oculomotorius. At the points, where in the section the optic vesicle is already reached, these cells cannot be detected.

An embryo of 6 mm. does not appreciably differ from the preceding stage. The nerves cannot be traced amongst the cells with perfect distinction. As best seen in a model, the cellular condensation extends first as a cylindrical body surrounding the n. abducens, and then is entered by the n. oculomotorius. In its further course it extends rostrally and towards the eye runs out into two processes.

On comparison of the projections of the embryos just described the gradual growth of the oculomotor musculature and nerve connection will be distinctly perceived. In the first described embryo of 7.6 mm. the beginning of the muscles innervated by the n. oculomotorius, and of the musc. rect. ext. innervated by the n. abducens, is already defined with perfect distinctness. The muscle plate for the n. oculomotorius begins to divide, still indistinctly, of course, into three plates, *i.e.*, future muscles.

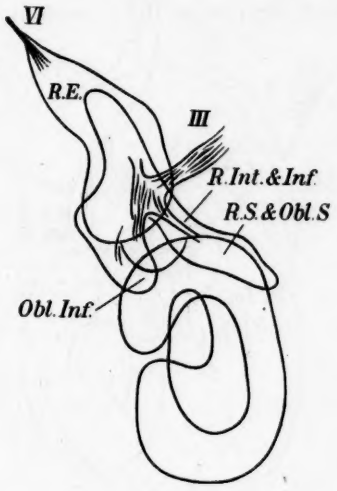


FIG. 2.

Embryo 8.4 mm. of the Spermophilus citillus

This division is suggested by three digitiform outgrowths extending for different distances towards the optic cup. Each of these outgrowths is entered by a branch of the n. oculomotorius, as can be seen on the general projection. The ligamentous division of the individual plates—muscles—cannot be observed yet with any particular distinction despite the mesoblastic elements issuing out of the surrounding connective tissue and reaching amongst the condensed cells.

If we now pass to a stage of 8.4 mm. the conditions we find do not essentially differ from those prevailing at the preceding stage. In point of growth, of course, the development of muscle plates has advanced so far that the digitiform processes of the plate,

originally common for the oculomotor musculature, are now divided into three plates, each of which is entered by a branch of the common oculomotor trunk, which first spreads into a considerable area and then gives off the individual branches out of this expanded part. The plate of the n. abducens has grown both long and wide. At this stage of development it is already possible to distinguish the foundation of the four ocular muscles:

1. The musc. rect. ext. from the n. abducens and
2. The musc. rect. sup.
3. The musc. rect. inf. and
4. The musc. rect. inter. from the n. oculomotorius.

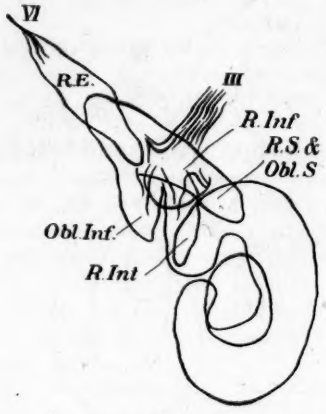


FIG. 3.

Embryo 9 mm. of the Spermophilus citillus.

These plates form, as it were, a basket on the posterior wall of the bulb. At this stage we must mention the peculiar conditions caused by the second branch of the n. trigeminus.

As stated above the foundation for the ocular musculature is situated in close connection with the ganglion of the fifth nerve, which, however, has nothing to do with the development of these muscles. However, at the stages of which I am treating we observe that the second branch of the fifth nerve also adjoins the very beginning of the muscle plates of the ocular musculature, and that around this branch there are also muscle cells which at some stages almost mingle with the cells of the eye muscles on the caudal pole. This, however, is a foundation for the masticatory musculature which develops in proximity to the eye muscles from

the second branch of the fifth nerve. Such proximity and contact are contingent on the structure of the future orbit which in some mammals has its fissure open wide, and through this the masticatory muscles pass. Now I dare suggest that it may be due just to this circumstance that some authors regarded the development of the m. rect. ext. as contingent on the fifth nerve.

At further larger stages we can especially observe the enlargement of the various processes, or, more exactly, the growing

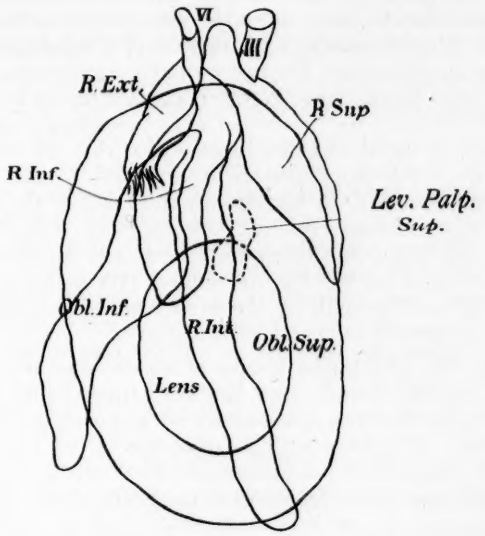


FIG. 4.

Embryo 10 mm. of the Spermophilus citillus.

of the various muscles towards the points of their attachment to the eye. The individual muscles are separated from one another by the intrusion of the mesoblastic cells, *i.e.*, the connective tissue. As soon as the n. trochlearis has grown to its full extent and joined the plate it is easily possible to distinguish the developed m. rect. ext., the cellular group for the musculature of the n. oculomotorius, and the m. obliqu. sup. which latter is derived from a portion of the oculomotor plate into which n. trochlearis has intruded on its anterior part. The growth of the m. rect. inf. is relatively very rapid. The plate for the

m. rect. int. and the m. obl. inf. remains in common for a fairly long period of time, and it is only after the connective tissue has intruded that the two begin to divide. The longest remaining together in one plate are the m. rect. sup., and the m. levat. palp. sup. from which the m. levat. palp. sup. is separted by the intrusion of the connective tissue. In an embryo of 10 mm. already all the muscles are developed as independent bundles, or they are spread flat—except for the division of the m. rect. sup. and the m. levat. palp. sup. At this stage the attachment of the musculature to the ocular wall is already distinctly defined, the various muscles are fixed on the connective tissue surrounding the eye, *i.e.*, on the developing sclera. Yet at none of these stages is it possible to speak of any definite development and deposition of musculature, and the final arrangement of the musculature can be observed only after the embryo has exceeded 2 cm. in length, and the facial region of the head has developed. For this the initial form and size of the developing eye are responsible. As I have stated in my earlier writings, in vertebrates the form of the eye undergoes very striking changes during development. The developing eye, at first decidedly oval with its longer axis dorso-ventral, grows into an oval form having its longer axis rostro-caudal, and the deposition and growth of the musculature corresponds with the successive phases of the developing eye.

In the *Sus* (the larger stages of which have been already dealt with by Kheil, Reuter, and Keibel) I have found the conditions, prevailing at the first developmental stage of the bulbar muscles, to coincide with those which I describe in the *Spermophilus*. I have not detected the slightest deviation either as regards locality of deposition or further development, even after the examination of a complete series of developmental stages. Moreover, I must add that no deviations can be detected even if I compare the developmental phase of the form of the eye to that of the muscles. The same must be stated in respect of the embryos which I could study only in incomplete series of several developmental stages (which again were studied only for the sake of comparison) of the *Vespertilio, Cuniculus,* and *Canis,* and also on a considerable number of the embryos of the *Mus* and *Cavia*. It may be stated that the development of muscles in higher mammals is governed by a common principle as regards the locality, deposition, and further growth.

The object of this study was to establish the very first beginning of the development of the orbital muscles in mammals. Further development and final arrangement of the musculature, as well as the first appearance of the fibrils of striated muscles, constitutes another department.

Summary

1. In mammals we find no structures whatever that would correspond to the head cavities in lower vertebrates.

2. The first beginnings for the development of the muscles which move the eye-ball appear as a condensation of mesoblastic cells between the carotid artery and jugular vein. The condensation is located in the region of the process for the upper jaw, medial to the ganglion trigemini, in the spot where the chorda dorsalis at the cephalic pole of the embryo terminates.

3. On the caudal side the abducens, and on the rostral side the oculomotorius, grow towards this condensation.

4. The condensation appears first as a cylindrical body medial to the optic vesicle, the anterior portion of the condensation being entered by the trochlearis.

5. Then this cylindrical body grows to form digitiform processes, and thus the primarily cylindrical cellular body divides into separate muscles, namely: the m. rect. ext., m. rect. sup., and m. obl. sup. Later on, during the growth of this cellular condensation, part of the cells separates from the cellular group innervated by the n. oculomotorius, but nevertheless remains common for the m. rect. inf. and m. obl. inferior. On the other side the process for the m. rect. int. separates from this cellular body.

The m. rectus super. and m. levat. palp. separate the last.

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TWO CASES OF TRAUMATIC KERATITIS IN THE NEW BORN*

BY

W. GORDON M. BYERS, M.D.

MONTREAL, CANADA

THE two following cases of traumatic keratitis in the new born are of special interest because of the findings in the corneae with the slit-lamp. The pathological changes underlying this disorder have been well understood since the classic article of Thomson and Buchanan[1], to whom we are also indebted for our first general description of the clinical picture; but up to the present time apparently no one has had the opportunity of studying these changes with the Gullstrand apparatus.

Case 1.—Mrs. . . . aged 30 years, a patient in the University Maternity Hospital under Dr. J. W. Duncan, was seized with labour pains at 6 p.m. on June 2, 1918. The following day, because of inefficient uterine contractions and irregularity of the foetal heart beats, the forceps was applied (mid-forceps case), and the child was delivered at 4.10 p.m. Great difficulty was encountered in this case owing to the fact that the head presented face to pubes. The pelvic measurements were normal.

The child was of normal size, and weighed eight pounds. It was slightly asphyxiated following delivery, but was readily resuscitated. There was partial paralysis of the seventh nerve on the left side.

I saw the baby on the afternoon of June 4, and noted the following conditions in the left eye: Marked swelling and tumefaction of both lids closing the palpebral fissure; very slight proptosis, and slight chemosis; vertical, and slightly oval band of interstitial infiltration or oedema of the cornea taking in the middle half of this structure for practically the whole of its diameter; corneal epithelium everywhere intact; anterior chamber and iris, hidden beneath the cloudiness, are plainly seen on both sides of the opacity, and appear quite normal. The right eye is unaffected.

During the next twenty-four hours the central haziness extended to practically the whole of the cornea; but, following this, subsidence took place. The paresis of the seventh nerve disappeared within a week of birth.

This spring, that is, after a lapse of six years, I again had the opportunity of examining this patient. She is now a sturdy girl with no suggestion of scarring about the face. The head is tipped

*Read before the American Ophthalmological Society, Hot Springs, Va., June, 1924.

noticeably to the right and down, and there is a convergent strabismus (markedly spastic) of the left eye. The excursion of the globe, however, is not impaired.

The left eyeball is of the same size as the right, and everywhere of normal appearance, except for the changes in the cornea. In this structure, by ordinary focal illumination (the opacity is not discernible with the naked eye), one sees a faint linear scar running across its entire breadth from eleven to five o'clock. The cicatrix appears uniformly nebulous—has no denser central streak—and measures about 2 mm. in breadth, except over the pupillary area where it is slightly broader and oval in form. The borders, one would say, are rather sharply defined.

With the slit-lamp (oc.2 obj. a^2); by diffuse illumination one sees the scar with great clearness; but though magnified, it is of the same form as with ordinary focal illumination. By retro-illumination, on the other hand, the cicatrix resolves itself into a cirrus-like opacity, denser in the centre, but fraying finely at the edges, especially on the temporal side. By direct lateral illumination, one sees no thickening of the cornea in the area involved; but one gets a snowy reflection from the deepest layers, and with a narrow bundle one can observe a stratum of greater density immediately at or on Descemet's membrane. There is, in fact, a thin layer of scar-tissue at the posterior surface of the cornea preventing examination in the reflecting zone. Pigment deposits are not discernible.

Pupil, 4 mm. (same as right), regular and active; fundus normal; V.=1/60 only; field of vision full. Retinoscopy shows an astigmatism as follows: -5.0D. cyl. 170°; but no improvement in sight can be got with glasses.

Case 2.—Mrs. . . . aged 28 years, was admitted to the Hebrew Maternity Hospital on January 9, 1924. Pelvic measurements; inter-spinous 23½ cm.; inter-cristal 25½ cm.; external-conjugate 19 cm. After twenty hours of labour, irregularity of the foetal heart developing, the child was delivered as a mid-forceps case at 11.40 p.m. At birth the child was greatly cyanosed and deeply asphyxiated.

The following afternoon (January 10) the case was seen by Dr. Rosenbaum who made the following notes: Marked ecchymosis of the left side of the face, lids, and forehead; general haziness of the left cornea, but no loss of epithelium, and no circum-corneal congestion; anterior chamber, of normal depth, shows a hyphaema 3-4 mm. in height; pupil slightly smaller than that of the right eye.

Three days later (January 13) the cornea was decidedly clearer, except for the pupillary area, which showed indefinite clouding.

The pupil had regained its normal width, and the blood in the chamber was also much less.

On January 23, 1924, Dr. Rosenbaum was kind enough to have the parents bring the case to see me at the Royal Victoria Hospital. All signs of swelling and ecchymosis had disappeared, as well as the hyphaema, and the eye was free from congestion. By focal illumination one saw a typical, but faint, linear opacity, about 4 mm. in breadth, running across the whole surface of the cornea, from 12.30 to 6.30 o'clock.

It seemed an absurd thing to attempt, but my colleagues were able to hold the sleepy baby at the slit-lamp, and to draw the lids apart so that I was able to see quite distinctly, and a number of times, a rupture of Descemet's membrane by retro-illumination.

On May 1, 1924 (almost four months after birth), I was enabled through the courtesy of Dr. Rosenbaum to see the baby for a second time, and to make the following fuller notes: Left eye: no congestion; pupil regular, active, and of the same size as the right; no changes in the iris, which is of a slaty colour; extremely faint, slightly oval scar, which occupies the middle third of the cornea over the pupillary area, and runs from 12.30 to 6.30 o'clock. On close inspection the opacity resolves itself into a central band with a long, relatively clear streak in its middle, and three other fine, wavy lines that stand out from the general haziness—one to the inner side, and two to the outer side, of the central band. The opacity to-day is much less pronounced than at the time of our first examination. The fundus is normal.

With the slit-lamp (oc. 2, obj. a^2), observations by direct lateral illumination are impossible; but by retro-illumination one again clearly sees the rupture in Descemet's membrane. To-day it shows a very slight double curve like the letter "S." It lies behind the centre of the scar and has the same general line of direction. Its borders are more glistening, and one gets the impression of rounded rather than square edges.

Comment

Thomson and Buchanan (*l.c.*) recognized three varieties of change in the cornea following lesions of this structure occurring during labour: (1) a diffuse opacity which is temporary; (2) a diffuse opacity indeterminate in position which is permanent; (3) an opacity which takes a linear form and is permanent.

The cases in the third class, under which the two just reported fall, were shown microscopically by these writers to be due to rupture of the posterior elastic lamina in association at times with rupture of the deep lamellae of the *substantia propria*; and they

held that a permanent scar remained in either case as a result of the subsequent formation of cicatricial tissue.

The findings with the slit-lamp of the rupture of Descemet's membrane and of the cicatricial tissue in my second case and in my first case respectively form interesting confirmation of the connection between those changes and the corneal opacity.

The rapid clarification of the cornea in the baby makes it probable, however, that complete disappearance of the nebula will take place at an early date. This outcome would be really more in accord with our clinical experience of simple wounds of the posterior elastic lamina. The point has a bearing upon prognosis.

In my first case, the fact that the opacity, apparently a compact band by ordinary illumination, resolved itself by retro-illumination into a much more widespread and quite irregular scarring, shows as one would expect that we cannot draw hard and fast lines between the cases in Class 1 and those in Class 2 of the Thomson and Buchanan classification.

In one of Thomson and Buchanan's cases, microscopical examination showed masses of pigment granules in the coagulated lymph upon the exposed corneal surface, and between the coils of a ruptured Descemet's membrane. In my second case, where the conditions were favourable, I could not discern pigment. But it has occurred to me that we may have in these birth injuries an explanation of some of the obscure cases of congenital spindle-shaped pigmentation of the cornea so ably described by Dr. Holloway[2] before this Society in 1910.

In the absence of ophthalmoscopic changes, the great reduction of central vision in my first patient must be attributed apparently to an amblyopia from disuse.

Illustrations of the linear scars in traumatic keratitis of the new born having been so well done by Thomson[3] and by James[4], there seemed to one to be no necessity for a further attempt in this direction.

Postscript

Since reading the foregoing paper at the meeting of the American Ophthalmological Society, June, 1924, I have had the opportunity of seeing a third case of the kind described through the kindness of my colleague, Dr. Fred Tooke.

The patient was a girl, aged six years. Forceps had been used at her birth. Following delivery the eyelids of the right side were greatly swollen and ecchymosed, and the eye could not be opened for a few days. There was an abrasion of the skin near the outer canthus, and another of the right side of the nose, about one-

half inch below the canthal ligament, where a bony depression can still be felt. One would infer from the statement of the mother in regard to the facial abrasions noticed in this patient immediately after birth that, while the lateral edges of the blade impinged above the points mentioned, the inner edge of the extremity of the blade exerted pressure upon the anterior surface of the cornea, causing rupture.

By diffuse illumination one saw a faint opacity of the cornea, roughly triangular in shape, which occupied the middle two-thirds of the pupillary area. The apex of the scar pointed to eleven o'clock. Its margins appeared sharply defined.

The origin and precise limits of the scar were, however, only revealed by retro-illumination by the slit-lamp. In this lighting one could see with great distinctness, and follow, a characteristic rupture of Descemet's membrane. The fine brilliant line demarcated without interruption an area, spindle-shaped with curved ends, that extended from the extreme periphery of the cornea at five o'clock to almost the extreme periphery at eleven o'clock.

By direct lateral illumination one could see that the opacity was mainly due to a thin layer of scar tissue in the deepest layer of the cornea, though minute opacities were distinguishable like flocculi in the overlying *substantia propria*. The dense central scar faded gradually toward the periphery.

The affected eye showed a myopia of approximately 15.0D. Central vision, however, was only 3/60 with correction. The other eye was normal with vision 6/6.

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CHANGES IN REFRACTION IN DIABETES MELLITUS

BY

W. STEWART DUKE-ELDER, M.A., B.Sc., M.B., F.R.C.S.

CLINICAL ASSISTANT, OPHTHALMOLOGICAL DEPARTMENT, ST. GEORGE'S HOSPITAL, LONDON

AMONG the rarer and more interesting of the ocular complications of diabetes mellitus are sudden changes in refraction. Both an increase and a decrease in the refractive power occur; myopia has long been recognised (occurring in 4 per cent. of cases, von Noorden [46]), hypermetropia is rarer. A considerable number of observers have reported cases of both conditions, and, on analysis, it would seem possible to co-ordinate their findings into a definite "law."—That the refractive power of the eye tends to vary directly as the sugar content of the blood; that is, there is a tendency to hypermetropia with decreased sugar, with increased sugar to myopia. This conclusion is strongly borne out by the study of three cases which have recently come under my observation in St. George's Hospital. In the first, refraction varied with mathematical precision with the blood-sugar content; the second showed hypermetropia following a decrease of sugar; while the third developed myopia as a terminal complication of a fatal diabetes.

CASE I

Showing Refraction varying with Blood-Sugar Content

W. J., male, aged 31 years. Previous health very good: led a healthy, active life, including four years service in France during the war. "For some years" had noticed his urine "muddy"; and in 1914 was turned down in an application for a life insurance policy. The case is, therefore, probably one of a mild symptomless diabetes of long standing, with, recently, an acute exacerbation. Unfortunately there was no record of his previous refraction, but he insisted that his sight has always been perfect—in any case it allowed him to follow his occupation as a clerk with perfect ease.

Eight weeks before admission to hospital he noticed he was easily fatigued, and three days before he came under observation he collapsed entirely. On admission he appeared on the verge of coma: he was emaciated, shrunken, and anxious, his breath smelt strongly of acetone, and his urine was loaded with sugar.

After the institution of rigorous diabetic treatment with insulin he improved dramatically. But the treatment would appear to have been too heroic; his blood-sugar temporarily fell below normal, he developed symptons of hypoglycaemia, and, quite suddenly, on the following day, he complained of dimness and mistiness of vision. On examination he was found to have developed a pronounced hypermetropic astigmatism.

The progress of the case can most easily be followed from the accompanying charts. The urine-sugar and acetone bodies (Fig. 1.) were estimated daily, the blood-sugar at somewhat longer intervals, and to co-ordinate the results several sugar tolerance tests were done, one of which is figured. (Fig. 4.) The nature of this curve incidentally proves that the patient was a true diabetic. The refractions were measured every third day, in each case under cycloplegia (Fig. 3.), and the appropriate lenses always brought his vision easily up to 6/5. During the whole period he was under observation the fundus and vessels remained normal, the media clear, the lens showed no trace of opacity, there was no spasm or paralysis of the external musculature, no esophoria, the visual fields remained unimpaired, and the tension normal.

Period A: 2/3/24 to 12/3/24.

The blood-sugar was rapidly reduced from an unknown height to 0.062 per cent. On 3/3/24 the refraction read:

R.V.: + 5.25D. + cyl. 0.75 ax. 150°.
L.V.: + 4.75D. + cyl. 0.75 ax. 30°.

Carbohydrates in the diet were increased and the insulin dosage lessened, and by 12/3/24 he was excreting 34.75 gms. of glucose per diem. During this period of sugar increase the refraction changed, as figured, to:

R.V.: + 2.0D. + cyl. 0.75 ax. 150°.
L.V.: + 3.0D. + cyl. 0.25 ax. 30°.

Period B: 13/3/24 to 24/3/24.

The sugar again was rapidly reduced by insulin and starvation to an excretive value of 2.62 gms. per diem. After this second fall a second "hypermetropic crisis" occurred. On 14/3/24 the refraction gave:

R.V.: + 3.25D. + cyl. 0.5 ax. 120°.
L.V.: + 3.25D. +. cyl. 0.75 ax. 60°.

The blood-sugar was thereafter allowed to increase, latterly by largely adding to the carbohydrate intake and withholding insulin, until it reached 0.293 per cent. with a urine excretion of 169.05 gms. per diem, during which time a decrease in hypermetropia occurred. Refraction on 24/3/24 gave -

R.V.: +1.25D. + cyl. 0.25 ax. 120°.
L.V.: +1.25D. + cyl. 0.25 ax. 60°.

Period C: 25/3/24 to 25/4/24.

A further reduction of sugar to 0.066 per cent. in the blood, and 1.55 gms. daily in the urine, caused hypermetropia again to appear: on 31/3/24:

R.V.: +2.0D. + cyl. 1.25 ax. 105°
L.V.: +2.5D. + cyl. 0.5 ax. 75°.

Thereafter his blood-sugar was kept approximately normal, *i.e.*, at slightly less than 1 per cent., and the refraction fell as charted until it reached a value of:

R.V.: +1.5D. + cyl. 0.25 ax. 105°.
L.V.: +1.5D. + cyl. 0.25 ax. 75°.

at which value it appeared to stabilize. By 24/4/24 his metabolic condition appeared to have become stable on a fairly liberal diet aided by 30 units of insulin daily, his eyes appeared to have reached what was presumably their normal, and he himself felt, and looked fit and well. He was therefore discharged provided with instructions as to diet and insulin dosage.

Period D: 9/5/24 to 19/5/24.

Fourteen days later he returned and proceeded to go through a fourth sugar-variation phase, on this occasion pathological, when before it had been experimental. A badly sterilized insulin syringe at home had given him a cellulitis of the left arm. This was treated, but by 14/5/24 the blood-sugar had risen to 0.292 per cent. and he was excreting 55.61 grms. in the urine per 24 hours. Meantime his refraction, which, when he came into hospital on the 9/5/24 was still at the previous (normal) value, tended towards myopia, and now read:

R.V.: +1.0 D. + cyl. 0.25 ax. 105°.
L.V.: +0.75D. + cyl. 0.5 ax. 75°.

Period E: 19/5/24 to 9/7/24.

The sugar remained high until 19/5/24, and the (comparative) myopia persisted. The arm then cleared up, the sugar fell to normal rapidly, and on the 24/5/24, when the blood-sugar value was 0.095 per cent., refraction showed a hypermetropia.

R.V.: + 2.25D. + cyl. 0.25 ax. 105°.
L.V.: + 2.0 D. + cyl. 0.5 ax. 75°

Thereafter he progressed steadily, and one week later was again discharged with a normal and stable blood-sugar, and a refraction once more reading:

R.V.: +1.5D. + cyl. 0.25 ax. 105°.
L.V.: +1.5D. + cyl. 0.25 ax. 75°.

This persisted until July 19th.

CASE II

Showing Hypermetropia with Sudden Decrease of Sugar

L. L., female, aged 37 years. Previous health good; diabetes had never been suspected. She gave a history of having been out-of-sorts and drowsy for one week, and of having vomited persistently

for one day; she had been without food of any kind for the previous forty-eight hours. On admission she was found to be in a state of semi-coma; she was very lethargic, her face was flushed, her breath smelt strongly of acetone, her urine was loaded with sugar and acetone-bodies, and her knee-jerks were absent. Her blood-sugar was found to be 0.493 per cent. Her eyes were in every respect perfectly normal, and her vision, as far as her state would permit of its being tested, was good. The refraction corresponded to that of glasses which were brought with her, obtained on prescription from an ophthalmic surgeon some two years previously, viz.:

R.V.: 0D. + cyl. 0.5 ax. 70°.
L.V.: +0.5D. — cyl. 1.5 ax. 160°.

Insulin in large doses was exhibited together with glucose by the mouth. Within the first twelve hours she had received 310 units, and on the following morning, with a blood-sugar value of 0.056 per cent. rising to 0.158 per cent., she was sitting up in bed comfortably eating breakfast. Her eyes were again examined in detail, and again found to be normal—fundus, media, lens, pupil reactions, accommodation, muscles, tension, etc. Unaided the V.A. gave:

R.V. = 6/5.
L.V. = 6/9.

and, with her glasses, both eyes reached 6/5.

Thereafter she was put on a diet of 1,500 calories (containing 74 gms. total glucose value) with a daily insulin dosage of 60 units falling gradually in 26 days to nil. Under this régime she remained perfectly well, her urine remained continuously sugar-free, and her blood-sugar at approximately the normal value of 0.1 per cent. The course of the case can be seen in figs. 5, 6 and 7.

On the morning of the third day after admission her knee-jerks returned (an unusual event), and, in the afternoon, on awaking from a sleep, she burst into tears saying she had "gone blind." On examination her eyes were found to be normal (tension, pupils, fundi, movements, etc.), but the refraction showed a remarkable degree of hypermetropia. She was put under atropin, and investigation showed:

R.V.: +6.0D. + cyl. 1.5 ax. 70° = 6/9.
L.V.: +5.5D. + cyl. 4.0 ax. 70° = 6/12.

—an addition of 9 dioptres in one axis.

She was kept atropinized during her stay in hospital, and her refraction steadily fell as charted in Fig. 7, until on 27/6/24 it had reached a value of:

R.V.: +1.0D. + cyl. 1.0 ax. 70° = 6/5.
L.V.: +2.0D. + cyl. 2.0 ax. 70° = 6/6.

She was then discharged with instructions as to diet. On reporting on 9/7/24 she was found to be still sugar-free, and her refraction, under cycloplegia, read:

R.V.: 0D. + cyl. 1.0 ax. 70° = 6/5.
L.V.: + 2.0D. − cyl. 1.0 ax. 160° = 6/5.

On 19/7/24, seven weeks after admission, the refraction had almost reached its normal value:

R.V.: 0D. +cyl. 0.5 ax. 70° = 6/5.
L.V.: + 1.0D. − cyl. 1.0 ax. 100° = 6/5.

CASE III

Showing Myopia with Increasing Sugar

R.L., female, age 21 years. This patient was not directly under my care, but I had the privilege of observing her latterly. She was a diabetic of old standing, and had been treated for diabetes in hospital in June, 1923, when she was put on insulin treatment. She was re-admitted in coma in December of the same year, and again, also in coma, on April 4th, 1924.

The condition of her eyes on the two previous occasions was noted as normal, objectively and subjectively. She had never worn glasses, and her people insisted that her eyesight had always been perfect until a short time before the last admission, when she complained of dimness of vision, which steadily increased. I saw her two days after admission: she was extremely drowsy and irritable, her blood-sugar was 0.483 per cent., and her eyes, while in every other respect normal (including the tension), showed a refractive value under atropin of R. — 14 D. sph. L. — 13 D. sph. In spite of all treatment her condition got progressively worse. On the eighth day, while the refraction remained stationary, the tension of the eyeball had fallen to nil; on palpation it felt soft like a water-bed. The next day the patient died.

Although in this case there is no record of any previous refraction, one would seem to be justified in assuming that there was some, probably a considerable, degree of terminal myopia.

These three cases thus show a gradual tendency to myopia with increasing sugar, and a definite hypermetropia, coming on suddenly, soon after a rapid decrease in the blood-sugar value.

A study of the literature has brought to light forty-five recorded cases illustrating this refractive change. These are tabulated below, the change in refraction, not the actual measurement, being given.

CASE I.

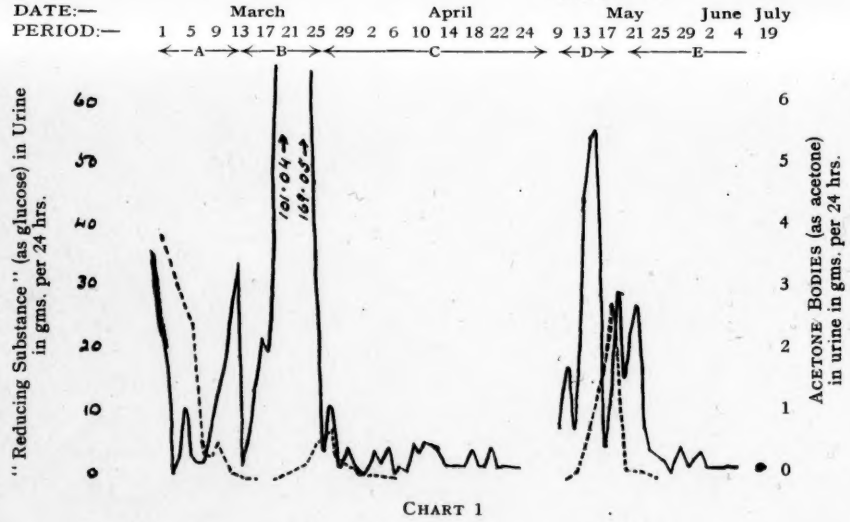

DATE:— March April May June July
PERIOD:— 1 5 9 13 17 21 25 29 2 6 10 14 18 22 24 9 13 17 21 25 29 2 4 19
A B C D E
"Reducing Substance" (as glucose) in Urine in gms. per 24 hrs.
Acetone Bodies (as acetone) in urine in gms. per 24 hrs.
101·04
169·05

CHART 1

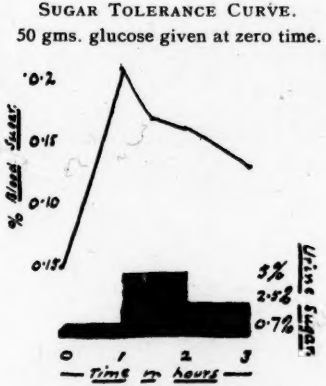


CHART 2

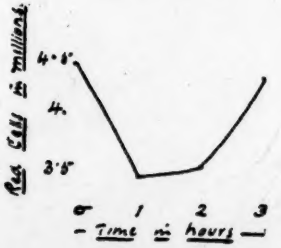


CHART 4

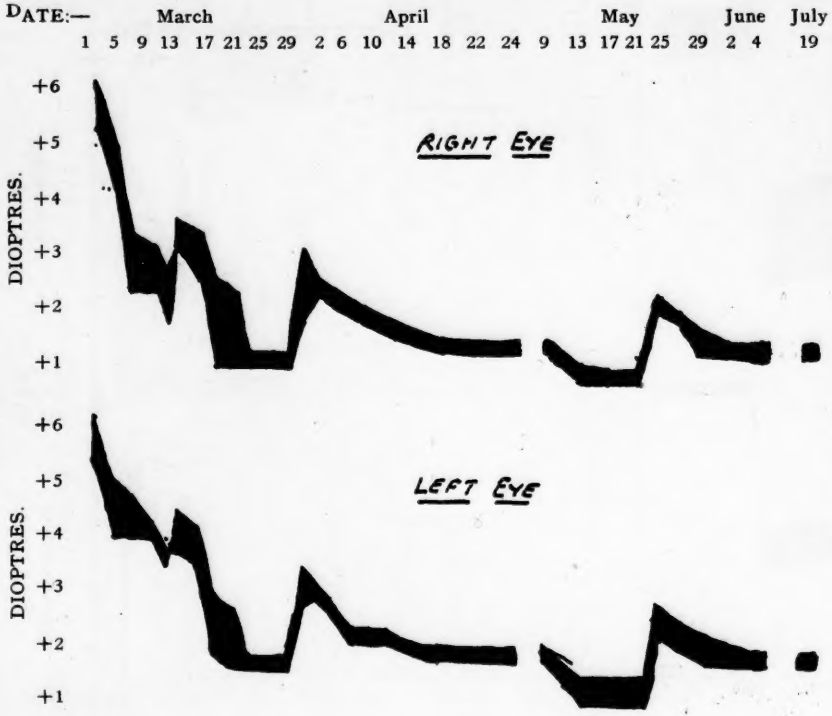


CHART 3

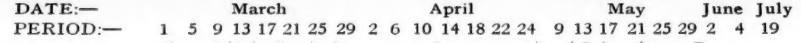
CHART 1 shows the sugar excretion per 24 hours. The figures are in gms. these giving a more accurate comparison than percentages, since the quantity of urine varies enormously with the sugar content. The readings are given daily. The dotted line indicates the excretion of acetone bodies: acetone, diacetic acid, and β. hydroxybutyric acid—also in gms.

CHART 2 indicates the variation in blood-sugar content expressed in percentages. To obtain uniformity the blood was in each case taken four hours after breakfast. These were estimated less frequently than the urine sugar, and thus the chart is less accurate. A comparison of the two is aided by a study of a sugar tolerance test (done while under insulin). 0.7 per cent. urine sugar represents approximately a secretion of 1.05 gms. per 24 hours; 5 per cent. of 7.5 gms.

CHART 3 shows the variation in refraction: the upper one of the right eye, the lower one of the left. The ordinates show dioptres of hypermetropia. The upper limit of each graph represents the axis of greatest curvature, the lower limit the least. Thus on the 3/3/24 the refraction of the right eye was $\frac{+6}{+5.25}$. On the 14/3/24 the axis of astigmatism changed. It will be observed that these curves vary inversely as the sugar curves.

CHART 4. The upper figure represents the reaction on the blood-content and urine of the ingestion of 50 gms. glucose, and the correlation between the two. The lower figure gives the variation in the number of red corpuscles per c.cm. of blood occurring at the same time. The blood-volume will vary in inverse proportion to this, *i.e.*, directly as the sugar-content.

CASE I.



CASE II.

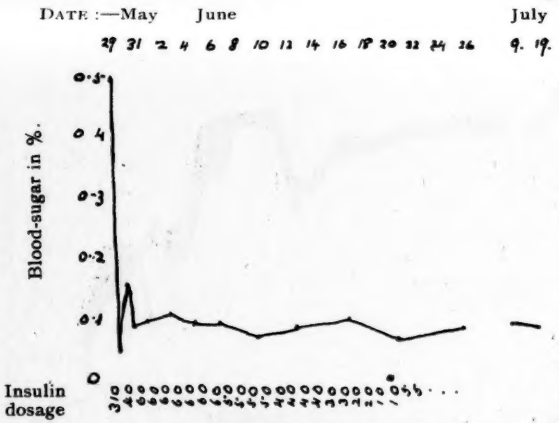


FIG. 5.—BLOOD-SUGAR CURVE

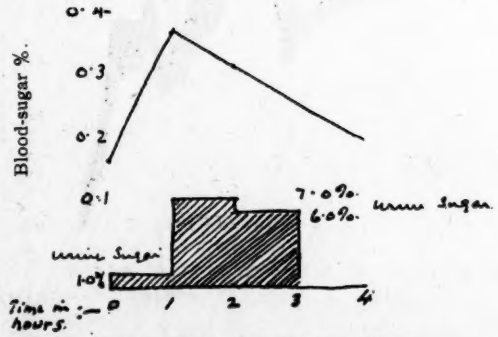
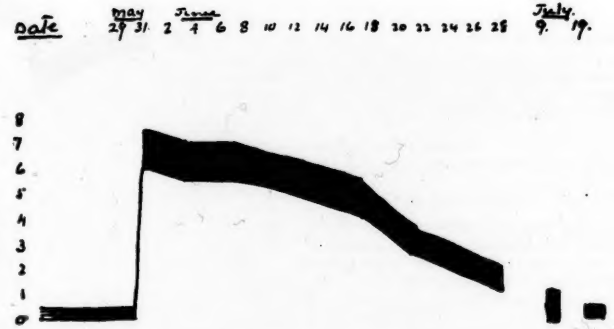
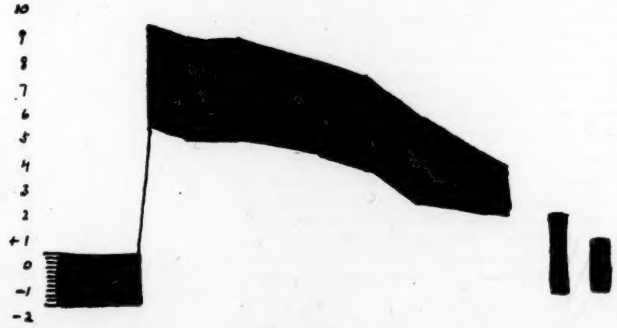


FIG.6.—SUGAR TOLERANCE CURVE
showing the typical diabetic characteristics

FIG. 7.
Curve of change of refraction in each eye.
(after Fig. 3.)



RIGHT EYE



LEFT EYE

GROUP I

MYOPIA WITH SUGAR INCREASE.

No.	Observer.	Sex and Age.	Change in Refraction.	Clinical Notes.
1.	Appenzeller, 1896	♀ 43.	−1	Returned to normal on treatment.
2.	Grimsdale, 1897	♀ 45.	R: − 3 D. − 1.25 cyl. (axis changed) L: − 3.75 D.	R. lens opacities centre. Loss of accommodation. Four weeks later coma and death.
3.	de Schweinitz, No. 1, 1897.	♂ 47.	− 1.75.	—
4.	de Schweinitz, No. 2, 1897.	♀ 20.	R: − 3 L: − 2.	Oedema choroid: vitreous opacities. Coma and death.
5.	Dujardin, 1900.	♀ 69.	− 5.	Sugar persisted on treatment: eyes remained myopic.
6.	Heine.	—	− 1.	Cataract.
7.	Neuberger, No. 1, 1903.	♀ 50.	R: − 1.5. L: − 2.	Returned to normal in three weeks, although sugar increased.
8.	Neuberger, No. 2, 1903.	♀ 48.	R: − 3.75. L: − 8.75.	Progressing cataract.
9.	Alexander, 1903.	♂ 57.	− 1.	Urine Sugar, 5.7 per cent. In 13 days sugar-free, and refraction normal.
10.	Landolt, 1904.	♀ 60?	?	Observed for several years. Each exacerbation of diabetes announced itself by a diminution of refraction.
11.	Jackson, 1905.	♀ 52.	− 1.75.	Soon died.
12.	Groenouw, 1907.	♀ 52.	− 1.	Three weeks' treatment, refraction normal.
13.	Wescott, 1911.	♀ 47.	R: − 1.5 + 0.25 cyl. (axis changed). L: − 1.25+0.25 cyl.	Urine sugar 5 per cent. Although sugar remained high, refraction tended to return to normal, adding + 1.25 D of Hm. in four months. Coma and death resulted.
14.	Elschnig.	♂ 14.	− 2.5.	Urine sugar, 7 per cent. + acetone. One month later death in coma.

DATE:—May June July

CASE II.

FIG. 7.

GROUP II

HYPERMETROPIA WITH SUGAR DECREASE

No.	Observer.	Sex and Age.	Change in Refraction.	Clinical Notes.
15.	Hirschberg, 1891.	♂ 48.	+sph. 1 + cyl. 40	Patient myopic: normal −9D. sph. Change occurred 14th day of treatment.
16.	Risley, No. 3, 1897.	?	+ 1.25 cyl. ax. 90°.	Appeared and disappeared with paresis of accommodation and esophoria. Due to change in corneal curvature.
17.	Gallus, 1906.	♂ 51.	R : + 1.5. L : + 2.	Occurred on second week of treatment. Normal in two months, when sugar-free.
18.	Lundsgaard, No. 1, 1907.	♀ ?	R : + 2. L : + 1.5.	Occurred suddenly on treatment. Normal in four months when sugar-free.
19.	Lundsgaard, No. 2, 1910.	♀ 50.	R : + 1.5 L : + 3.	Occurred 15 days after treatment commenced. During first 12 days urine sugar reduced from 6.2 per cent. to 0.2 per cent.
20.	Knapp, No. 2, 1909.	♂ 48.	+ 1.	Paralysis of accommodation. Occurred a few weeks after treatment began. Normal in four weeks when sugar-free.
21.	Ellis, No. 1, 1911.	♀ 40.	+ 4.5.	Occurred two to three weeks after treatment began. Returned towards normal slowly with the appearance of a small cylinder. In three months when sugar was still 2.5 per cent. R : + 2.25. L : + 1.37.
22.	Ellis, No. 4.	?	+ 1.5 D. + 0.5 cyl.	Occurred on treatment one week.

GROUP II.—*continued.*

HYPERMETROPIA WITH SUGAR DECREASE

No.	Observer.	Sex and Age.	Change in Refraction.	Clinical Notes.
23.	Zentmayer, 1912.	♀ 58.	R : + 2.5 + 0.25. L : + 2.5 + 0.5.	Occurred 10 days after treatment started. Sugar reduced from 5 per cent. to nil. In two months refraction almost normal : R : 0.5 D. L : 0.75 D.
24.	Post, 1917.	♀ 59.	R : + 0.75. L : + 1	? on treatment. Normal in four months.
25.	Spalding, 1923.	♀ 29.	R : 3.75 + 0.75 cyl. L : 3.5 + 0.25 cyl.	Occurred on second day's starvation.
26.	Roberts, No. 1, 1923.	♂ 51.	+ 1.75.	Occurred two weeks after treatment began. Sugar reduced from 7 per cent. to nil in four weeks. Normal in six months.
27.	Roberts, No. 2, 1923.	♀ 54.	R : Nil. L : + 1.75.	Occurred after one week's dieting. Five weeks later returned to normal with a fall of 1.25 D + 0.25 cyl. in R.E.
28.	Roberts, No. 4, 1923.	♀ 55.	+ 4 + 0.25 cyl.	Occurred on sugar falling from 10 per cent. to nil. Normal in three months. Later, on restricted diet, vision blurs; on eating carbohydrate, vision clears up.
29.	Roberts, No. 5, 1923.	♀ 66.	R : + 3.25. L : + 2.75.	On treatment four weeks. Sugar reduced from 7 per cent. Normal in three weeks.
30.	Elschnig, No. 1, 1923.	♀ 47.	R : + 2. L : no change.	Occurred 14 days after treatment. Three months later was returning to normal. The L.E. had had a previous cataract extraction. It remained constant + 9.5 D sph. + 4 cyl. = 6/12.

GROUP III

SHOWING BOTH CHANGES

No.	Observer.	Sex and Age.	Change in Refraction.	Clinical Notes.
31.	Risley, No. 1, 1897.	♀ 49.	(a) R : + 2.25 +1.25 cyl. L : + 3.25 + 0.75 cyl. (b) R : −3.75. L : −4.5.	(a) Occurred on commencement of treatment : i.e., sugar diminished. (b) 25 days later : sugar increased.
32.	Risley, No. 2	♂ 74.	(a) R : +1. L : +0.75. (b) R : −1. L : +0.75.	(a) On treatment 14 days : sugar a trace. (b) One month later : sugar 4.2 per cent.
33.	Carpenter, 1902.	♀ 51.	(a) R : + 1.75 + 0.5 cyl. L : + 2.5 + 0.5 cyl. (b) R : −2.25 L : −3.	(a) On treatment four weeks. (b) Patient left off diet.
34.	Gould, 1907.	♂ 58.	(a) R : −0.63 D. L : −0.63D. −0.25 cyl. (axis changed). (b) R : +1.25 +0.13 cyl. L : + 1.38 + 0.13 cyl. (axis changed).	(a) Untreated. (b) On treatment.
35.	Knapp, No. 1, 1909.	♀ 47.	(a) −2. (b) R : +3. L : +2.5 (c) R: −0.5. L: −0.25.	(a) Untreated. (b) After 14 days' treatment when sugar-free. (c) After 14 days' sugar-free (returning to normal).
36.	Lundsgaard, No. 3, 1910.	♀ 50.	(a) R : +1. L: +0.5. (b) R : −2.5. L: −2.	(a) Sugar reduced from 6 per cent. to 0.85 per cent. (b) Sugar increasing.
37.	Lundsgaard, No. 4, 1919.	♂ 41.	(a) +2.5. (b) −2. (c) −0.5.	(a) Ten days' treatment : sugar, urine nil ; blood 0.120 per cent. (b) Six days later : sugar, urine 2.4 per cent. ; blood 0.217 per cent. (c) Six days later : sugar, urine 0.3 per cent. ; blood 0.180 per cent.
38.	Ellis, No. 3, 1911.	♀ 65.	(a) R : + 2.5 + 1.25 cyl. L : + 2.5 + 0.75 cyl. (axis changed). (b) R : − 1.75 − 1.0 cyl. L : − 1.75 − 0.75 cyl. (axis changed).	(a) On treatment, four weeks. (b) Five months later, treatment neglected.

GROUP IV

ANOMALOUS CASES, AND CASES LACKING SUGAR DATA

No.	Observer.	Sex and Age.	Change in Refraction.	Clinical Notes.
39.	Horner, 1873.	♀ 55.	(a) + 2. (b) − 2.	No mydriatic used. No sugar data.
40.	Cohen, 1878.	♀ 68.	+ 1.5.	No mydriatic used. No sugar data.
41.	Doyne, 1897.	♂ 40.	(a) + 3. (b) − 2.25.	Was +3 D. when had sugar + was 0.75 D. when sugar-free some months later; but no data of condition before or during the interval.
42.	Sourdille, 1900.	♂ 53.	+ 2 D.	Developed suddenly: normal in eight days when sugar-free. No mydriatic used. Syphilis +. No definite sugar data.
43.	Lichtenstein, 1906.	♂ 17.	+ 2.	Apparently a Hm. with sugar +. It lasted 14 days, and returned to normal some weeks later. Accommodation was paralysed, adding + 13 D. (sic) for near vision. No definite sugar data.
44.	Ellis, No. 2, 1911.	♀ 48.	(a) R: − 0.25 cyl. (axis changed). L: − 0.25 D. (b) R: − 1.75. L: − 2.0. (c) R: + 0.25. L: + 0.5 + 0.25 cyl.	(a) Measured when urine sugar was 4.5 per cent. (b) Three months later, when sugar-free, i.e., an apparent myopia with sugar decrease; but estimations only at prolonged intervals. (c) Two months later.
45.	Roberts, No. 3, 1923.	♀ 53.	+ 1.5 + 0.25 cyl.	Normal in four months. Left chorio-retinitis. Right swollen disc. Patient almost blind. No definite sugar data.

To account for this refractive change a great number of theories have at various times been advanced, including the suggestion that refractive errors are a causal factor in the aetiology of diabetes. The possible causes may be considered briefly: most can as briefly be dismissed.

I. DYNAMIC.—Dependent on a neuro-muscular mechanism

1. Affecting the internal musculature.

Paresis of accommodation, allowing a latent hypermetropia to become manifest, in part or in whole (Groenouw, Schmidt-Rimpler[12]).

This does occur in some cases (Nos. 16, 20, 43); it may account for part or the whole of the change in some (Nos. 39, 40, 42); but, applied to the majority, the theory breaks down.

Thus: (*a*) The range of accommodation has repeatedly been found to be normal.

(*b*) A mydriatic eliminates its influence.

(*c*) The ages of many of the cases are beyond that during which accommodation is active (Nos. 5, 29, 38, 40).

(*d*) Many of the cases developed a degree of hypermetropia greater than the range of accommodation would allow (Nos. 5, 8).

(*e*) It cannot account at all for the occurrence of myopia.

2. Affecting the external musculature (Risley[16]).

In Case 16, the hypermetropic astigmatism, apparently due to a change in corneal curvature, which appeared and disappeared with esophoria, presumably depended on a spasm of the laterally acting muscles. But this would appear to be isolated, and, when it has been looked for specially in several cases, it has been found absent.

II. STATIC.—Dependent on a physico-chemical mechanism

1. Affecting the coats of the eye.

(i) A shortening of the antero-posterior axis due to a shrinkage of the eye from loss of fluid in the vitreous. (Sourdille[42]: Lichtenstein[43].)

This undoubtedly does occur in advanced cases, as a terminal event in diabêtes when the tissues generally have become dehydrated. It is well known clinically as the "soft eye" of diabetic coma.

But (*a*) If it occured at all in milder cases the result would be a hypermetropia with increasing sugar. The theory was advanced to explain the anomalous cases already dealt with.

(*b*) The tension has been repeatedly measured and found to be normal.

(ii) A lengthening of the antero-posterior axis, due to softening and yielding of the sclera, the underlying pathology being a diffuse oedematous condition of the choroid. (de Schweinitz and Kneis[4].)

But (*a*) although this might have occurred in one case (No. 4), when the diabetes was very severe, and the eyes otherwise gravely affected, in milder cases the theory is not tenable, and clinically no such choroiditis occurs.

(*b*) Such a theory cannot account for an initial hypermetropia.

2. Affecting the media generally—an index change. A general alteration in the index of refraction due to the presence of sugar and other metabolites, rendering the refraction greater, and thus inducing myopia as diabetes progressed, rendering it less, and inducing hypermetropia with a decrease in the severity of the disease. (Landolt and de Wecker[48].)

This seems delightfully simple, but:

(*a*) The blood-volume normally contains a very small actual amount of sugar. Recent work [49] has placed the blood-volume at 1/13 of the body weight. With an average blood-sugar value of 0.1 per cent., an average man of 65 kilos. will have normally circulating in the blood 5 gms. of sugar. The highest diabetic value found in a very extensive investigation [50] was 15 gms. of total sugar, or 10.75 gms. of plasma sugar. The increase, therefore, at any time is small, in the eye itself extremely small—too small to bring about *per se* a refractive change of 8.75 (No. 8) or 9 dioptres (Case II). A solution of sugar must be concentrated by 20 per cent. to bring about a change in its refractive power of 1 dioptre, and thus an increase of 1,000 gms. per dioptre would be necessitated.

(*b*) By this theory it is difficult to imagine why the refraction of every diabetic should not change. The phenomenon is comparatively rare. It would also necessitate a gradual rise and fall in refractive power, varying exactly with the sugar content; whereas the change, especially the hypermetropic change, is usually sudden.

(*c*) It cannot account for an astigmatic change, which occurs in 36 per cent. of cases in the series.

3. Affecting the lens. A lenticular change involving one or other, or both, of the following factors:

(i) A generalized change of mass and density, accompanied by a deformation of the anterior surface.

(ii) A change in the size and density of the cortical layer relative to the central nucleus (Grimsdale[27]), making the lens, for example in the hypermetropic change, a more uniform structure, tending to the infantile form, with a diminution of refractive power.

There would appear to be at least six considerations pointing to the lens as being largely the causal agent in the phenomenon.

1. 81 per cent. of the cases in the series occurred between the ages of 40 and 60 years, the period of lenticular instability.

2. Elschnig[30] found that while one eye underwent changes, the other, on which a cataract extraction had been done, remained stationary. This is the only instance where one eye alone has been affected, and it would thus seem that the presence of the lens is necessary for the phenomenon to occur.

3. Spalding[51] has noticed "a shrinkage and flattening of the lens."

4. The occurrence of astigmatic changes (in 36 per cent. of cases) suggests deformation of the lens as the origin.

5. It is conceivable to suppose that the character of this tissue and its mode of nourishment make it resistant to change; it is sluggish physiologically and pathologically. In the majority of diabetics changes do not occur. In some cases, after resisting osmotic forces for some time, it suddenly yields: this would account for what appears to be a very constant phenomenon and one somewhat difficult to explain. The sudden refractive change occurring some time after the sugar-value has fallen, while it also accounts for the rarity of the condition.

6. The lens in diabetes is unstable. The occurrence of cataract is common, and an opacity can readily be produced experimentally by sugar infusion in the frog[16]. Too close a parallelism should not, however, be drawn. It has been suggested that the myopia is comparable to that occurring prior to the sclerosing process of senile cataract, but this seems unlikely, in that the change is frequently sudden, there is a rapid improvement on treatment, lenticular opacities were detected in only 8.8 per cent. of the series, and such an analogy leaves no place for the incidence of an initial hypermetropia.

It seems most reasonable to explain these changes on the hypothesis that they are due to osmotic influences consequent on the disturbance of the water-balance of the body that forms so large a part in the general metabolic upset known clinically as diabetes. The conservation of normality in the constitution of the blood would appear to be one of the first and most urgent aims of the body-mechanism. A very small deviation from normal suffices to derange the nice equilibrium on which the processes of life depend: if there is any variation, the tendency is, at whatever cost, to keep it normal. As the blood-sugar increases the attempt is made, first, by the withdrawal, by osmosis, of fluid from the tissues, to dilute the sugar-laden blood, and secondly, to increase sugar excretion via the kidneys—(hence polyuria as a symptom). The net result of these two processes is to dehydrate the body, a

catastrophe which is usually averted by a largely increased fluid intake—(hence polydipsia as a symptom). Pushed to extremes, however, the body is drained of its fluids, the eye shares in the general dehydration, by extraction of fluid its tension falls, the soft eye of diabetic coma results, and death usually supervenes. This has been demonstrated experimentally by Hertel[47] by sugar infusion in animals, and more recently corroborated in America[52]. Hertel found that such an eye contained on evaporation less fluid than normal; that the process was a purely physical one was shown in that the same phenomenon occurred on the infusion of neutral salts as chlorides, sulphates, phosphates, and carbonates; and that it was osmotical, in that the electrical conductivity of the eye was increased, demonstrating an increase in molecular concentration.

With a raised sugar we have thus a dilution of the blood by tissue fluids, and an increased excretion from the blood of a concentrated fluid with a high salt content, and a high osmotic pressure (urine), which is replaced volumetrically by drinking (practically) water. These together produce:

1. An increase in blood-volume,
2. A decrease in blood-salt content,

both of which contribute to a lowering of osmotic pressure.

The change of volume may be explained thus: In health, the fluid traffic is regulated by the effective osmotic pressure of the blood over the lymph, due to the colloids of the former (20 to 30 mm. Hg.), inducing a flow from the tissues to the blood stream, compensating for the amount lost by the filtration effected mechanically by the blood-pressure. In diabetes, the osmotic pressure is further raised by the presence of increased crystalloid (glucose); an effective increase over filtration pressure is thus established, and fluid in excess flows from the tissues to the blood, to be replaced, of course, by increased intake. The change in volume was compared in both cases (1) and (2) before and after changes in the sugar concentration were brought about, and the above reasoning was confirmed. The investigation was made by determinations of the red cell count and the haemoglobin percentage, for, since the number of the red cells does not change, and the fluid bulk is made up by a fluid lacking in haemoglobin, while these values remain constant absolutely, they undergo a percentage change relative to the volume. Thus after the exhibition of 50 gms., glucose to case (1), the count fell by about a million per c.mm. during the first hour (Fig. 4) and rose again as seen in the chart, and the haemoglobin fell from 90 per cent. to 70 per cent. As the blood-volume (other things being equal) varies inversely as the red cell count and the haemoglobin, it is seen that it varies directly with its sugar content.

To determine the effect of sugar variation on salt content, the total chlorides in the blood were estimated by Volhard's method

before and after the occurrence of changes in cases 1 and 2. Thus in case 1, when the blood sugar was high on 25/3/24, the salt content was 0.49 per cent.; after the sudden fall it was 0.59 per cent. This lowering of the salt content in diabetes has been observed by others: Schmidt[53], Meyers[54], etc.; and MacLean[55], estimating the chlorides in pathological conditions, found them lowest in diabetes, the decrease being due to a lowering of the normal chloride threshold. This is explained by the tendency for an excess in one crystalloid (glucose) to be compensated for by a depreciation in another (salt). That the salt-change will exert more osmotic disturbance, is evident from its smaller molecule and its ionization, since osmotic pressure varies as the number of molecules, and if these molecules are dissociated electrically, each fraction acts as an element equivalent osmotically to a molecule. It might be thought that the higher osmotic pressure established by glucose which causes the increase in volume, and the lower pressure entailed by decreased salt, were mutually antagonistic: whereas they are complementary. Owing to its great diffusibility, salt equilibrium will be quickly established between the blood and tissue fluids. A fall in blood-salt thus demands a coincident fall in general fluid-salt. The larger sugar molecule diffuses more slowly, and therefore, while the salt equilibrium throughout is " set " at a lower level, the higher effective osmotic pressure in the blood due to excess crystalloid still is able relatively to effect changes in volume; thus on both counts, the osmotic pressure of the fluids bathing the lens is lowered.

Again another factor may enter into play. Applying the colloid chemical theory of absorption by protoplasm[56], it is probable that, in the increased PH of diabetic acidosis, with the subsequent buffering of the blood, some salt is transferred from an osmotically active state in the plasma to inactivity in the corpuscles, rendering the plasma still more dilute, and its osmotic pressure still less.

As the sugar content rises, therefore, the molecular concentration of the blood and tissue fluids falls. The lens, which we have already assumed to be peculiarly resistant to change, remains *in statu quo*, and is thus bathed by a fluid at a tension lower than its own molecular state; osmotic forces are set up, and to obtain equilibrium, fluid flows from the lower level to the higher, that is, into the lens, deforming it, and making the eye myopic. The increase of refractive power may be accentuated by a decrease in the index of refraction of the cortical layers, as opposed to the harder and more resistant nucleus, by dilution, thereby making the index of the whole lens less uniform, and therefore of a higher refractive power.

Conversely, on a fall of blood-sugar, a reverse osmotic change occurs, and some degree of hypermetropia is produced by the

transference of fluid from the lens to the surrounding tissues and blood, whose osmotic pressure is now raised.

Cataract is common in diabetes. It would seem probable that its incidence may be partly accounted for by the ready degeneration of a lens previously weakened by the long continued strain of a deforming force, with the additional element of increased fluid traffic. Diabetes is a metabolic dyscrasia, notorious for the many complications it brings in its train — complications ranging in diversity and in severity from crops of boils to mass gangrene, from a peripheral neuritis, whose only clinical manifestation is a lost knee-jerk, to a widespread and profound paralysis. In the eye, the element of infection is seen in diabetic iritis: nerve involvement is exemplified in paralysis of accommodation, in retrobulbar neuritis, and in retinitis. The retina, like the lens, is nourished indirectly, and is, therefore, peculiarly susceptible to osmotic influence; and in nephritis, where again the water-and-salt balance is out of joint, the retina is affected.

Can it not be that these are all due to the same fundamental cause? The reaction between host-organism and invading micro-organism is probably a question of the physico-chemistry of adsorption compounds, the excitation process of nerve, a matter of the diffusion of ions through semi-permeable membranes. It requires but a slight disturbance of osmotic forces to upset the nicely balanced equilibrium of the ionic and colloid systems on which the mechanism of life depends. At the least, reasoning thus, we approach more nearly the ideal concept, whereby the body, both in health and in disease, is an electro-chemical mechanism governed by purely physical laws, and get further removed from vitalistic views of normal and morbid processes, which are merely a dishonest confession of ignorance, are never the simplest, and are always sterile.

Conclusions. I suggest that:

1. In diabetes sudden changes of refraction occur. In the course of the disease with high or increasing sugar, myopia tends to occur: with decreasing sugar, and, therefore, usually just after the onset of energetic treatment, hypermetropia tends to occur.

2. The phenomenon is due to osmotic processes involving the lens, caused by a variation of the molecular concentration of the blood and tissue-fluids with the sugar content.

3. In both cases the tendency is to return to normal: in the first, under general diabetic treatment; in the second, on the establishment of metabolic stability. Provided the diabetic element can be successfully dealt with, invariably a good prognosis can be give n The patient can be assured that his eyes will return to norm a within a few weeks and that his vision will be unimpaired.

4. In consequence of the general use of insulin, it is anticipated that the rarer condition of hypermetropia will become more common.

I am indebted to Dr. James Torrens for permission to publish the first case; to Dr. James Collier for the latter two; to Mr. R. R. James for his helpful and encouraging interest in the investigation; and to Mr. W. G. Laws for his kindness in supplying me with the notes of his two cases, and for his permission to add a note on them to this paper.

Since writing this paper, Mr. W. G. Laws has very kindly given me notes of two unpublished cases which have come under his observation where hypermetropia developed after the appearance of diabetes. Both patients were middle-aged women; the onset of hypermetropia was sudden, and occurred soon after the institution of treatment. In one the hypermetropia amounted to 4D, and in the other to 2D. In both the refraction returned to emmetropia when the diabetes was kept in check by treatment. In the first case a strict diet was instituted; the second case was given insulin, but not apparently in 1922 when the diabetes was first diagnosed and when the development of hypermetropia took place.

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ANNOTATIONS

The Visual Requirements of Motor Drivers

In view of the fact that new legislation on the issue of motor drivers' licences is not unlikely in the immediate future it may be advisable to remind our readers that the Council of British Ophthalmologists issued a report in 1920 on the subject of visual requirements in motor drivers. This report, in which the whole ground is covered, was published in this journal (Vol. IV, 1920), and may still be obtained from the Council's secretary. The American Medical Association has recently published a report with recommendations read before its Ophthalmological Section (*Jl. of Amer. Med. Assoc.*, Dec. 27, 1924) which does not confine itself to visual tests but deals with all physical defects in a manner that would certainly reduce the number of drivers and add to the incomes of the certifying physicians at the expense of those who were granted licences. Their recommendations are as follow:

"1. Every individual driving a motor vehicle, private, public or commercial, of the self-propelled type (automotive) shall be required to present to the motor licensing board in each state a certificate from a reputable physician (the standard of such medical fitness shall be licensure to practice medicine or surgery) in which the following points are certified:

(*a*) The applicant has no disqualifying defects of either legs or arms (anatomic or functional loss of hand, arm, foot or leg).

(*b*) The applicant has vision of at least 20/50 in one eye and vision of at least 20/100 in the other eye with or without glasses.

(*c*) Double vision shall disqualify.

(*d*) All candidates must be able to hear a low spoken voice at 5 feet.

(*e*) The mentality of the applicant must be adequate and the heart's action reasonably healthy.

Examiners shall be recompensed by a fee paid by each applicant, the amount of such fee being determined by the motor boards in each state. The licence must be renewed every year, but the medical certificate must be made anew every three years, provided that the applicant makes a sworn statement each intervening year that, to the best of his or her knowledge, there has been no change in his or her physical condition, so far as the certificate applies, since the time of the last examination."

The report goes on to point out that while it is desirable that all these tests should be made by specialists in the particular parts of the certificate such a course would be impracticable. It also adds that colour vision testing would result in a rejection of 3 per cent. of males and so would speedily lead to the popular rejection of all tests. We rather fancy that the same effect is not unlikely to follow the adoption of the rest of their scheme in its entirety. The *British Medical Journal* for January 24, 1925, has a leading article dealing with this report. This article says: "The report was drawn up evidently by men who were earnest in the cause of public safety and experts in their own branch of practice. On the other hand it would appear that none of them can have been expert on the other side of the question—motor driving; and their earnestness would seem to have led them astray from the paths of common sense. Such men, when faced with a difficulty, are always liable to shirk the issue by calling on the State to increase bureaucratic control, regardless of arguments on the possible futility and costliness of the procedure."

There can, unfortunately, be no question of the regrettable increase in accidents in which mechanically propelled vehicles are concerned, but in how many of these is there any evidence that apart from alcohol and "neurasthenia" any of these are due to a physical condition on the part of the driver which would be excluded by any such tests as are likely to be applied in the filling up of such a certificate as that suggested by the American Committee? The chief cause of the increase of accidents is undoubtedly to be sought in the increase in the number of motors, not in an increase in the physical defects of their drivers. Paradoxical as it may sound we rather incline to the view that the driver with some moderate physical defect of which he is aware, such as slight deafness, is less, rather than more, liable to avoidable accidents, inasmuch as he does not trust entirely to hearing a horn at a blind corner but goes more carefully.

A reference to the report of the Council of British Ophthalmologists already referred to will recall the fact that they drew a sound distinction between the driver of his own car and the professional driver. The former can choose his route, his own

time and the pace at which he goes; the latter cannot do so. It is certainly essential that men who are to drive for long periods regardless of the weather or traffic conditions should be picked men. All motorists appreciate the skill of the London omnibus driver. He has to go through searching practical tests by experts. All motorists dread the Ford delivery van with the youth in charge who goes through no tests whatever. In a recent number of *The Times* there is a short account of the tests for motor drivers now being used in Paris. These are certainly of great value but call for very expert examiners. The three chief demands kept in view have been: (1) Power to attend rapidly to the movements of vehicles and persons; (2) ability to heed sounds coming from different quarters; (3) capacity to react effectively and quickly to such stimuli. Further, muscular force and "fatigability" are tested, and also rapidity of perceptions. Such tests are, of course, quite ideal, but without the resources and skilled workers of a regular anthropometrical laboratory of no practical value. In this note in *The Times* it is stated that at Barcelona it has been found that the frequency of accidents among 35 drivers who did poorly at these tests was 300 per cent. higher than among 45 drivers who did well at them. Either the frequency of accident at Barcelona must be a little out of the ordinary or this type of statistics must be of the kind that enabled Mark Twain to prove that the Mississippi had in quite recent times stuck out for hundreds of miles like a fishing rod into the gulf of Mexico.

The solution offered by the *British Medical Journal* has much to recommend it. "It might not be a bad thing, however, to require from every applicant for the issue or renewal of a driving licence a signed declaration that to the best of his belief he has no defect or disease which makes him unfitted to drive a car or motor-cycle. This would indicate upon whose shoulders the legal burden rested." We may add also that it would eliminate the plea of nervous shock, plus alcohol as an excuse in case of an accident.

Psicaine

Two papers on this new local anaesthetic are published in the *British Medical Journal* of January 2, 1925. The first is by Dr. Copeland, Ernest Hart Memorial Scholar of the British Medical Association. Psicaine is an optically active isomer of cocain which has been prepared by Willstätter in collaboration with Merck of Darmstadt. In Germany the new drug is twice as expensive as cocain; it is said to have double the anaesthetic power, half the toxicity, and to be free from addiction properties. It is

freely soluble in cold water up to 25 per cent., and it is not precipitated by saline solution, Ringer, nor by proteins.

Copeland finds that on the cornea psicaine has approximately one sixteenth the anaesthetic power of cocain; on the other hand a 0.5 per cent. solution of cocain and an 8 per cent. solution of psicaine are equally effective in giving complete anaesthesia averaging thirteen minutes under the same experimental conditions. Psicaine causes a moderate dilatation of the pupil in rabbits which lasts for twenty-four hours; a slight vascular congestion, but no drying of the cornea. Three instructive tables are given. The first shows the effects of local anaesthetics on nerve fibres, using: (1) the vagus, cardiac fibres; and (2) the anterior crural, sensory fibres. Comparisons are drawn between cocain hydrochloride, tutocain, psicaine and novocain. The second table shows the minimum lethal doses compared in the rabbit, guinea-pig and mouse for cocain, psicaine and novocain. The third table shows the minimum convulsive dose of these three drugs for rabbits, employing intravenous and subcutaneous injection. In rabbits psicaine injected subcutaneously was found to be more toxic than cocain; while intravenously the two drugs are of almost equal toxicity. In frogs, psicaine is a more powerful stimulant than cocain; the irritant effect of psicaine is less than that of cocain. The new drug has relatively little effect on blood-vessels; but in the cat, the mucous membrane became markedly swollen after an application of 5 per cent. psicaine by the author's perfusion method.

Copeland has been unable to verify the claims for efficiency and low toxicity; and states his opinion that if his experimental results are confirmed, psicaine is unlikely to be of use as a cocain substitute.

The second paper is by Dr. E. Watson Williams of Bristol. He deals with the subject from the clinical side in the human being in cases of nasal surgery. His conclusions are as follow:

"1. Psicaine is the acid tartrate of an artificial isomer of cocain. It is soluble in water, giving a definitely acid solution. The solution is not harmed by brief boiling. It requires the addition of salicylic acid to prevent the growth of moulds.

2. Used in the nose, 7 per cent. psicaine solution gives anaesthesia identical with that produced by 5 per cent. cocain hydrochloride solution. The anaesthetic value of the new alkaloid is the same as that of natural cocain.

3. The same "shrinkage" as is seen with cocain is found in using psicaine.

4. The experimental toxicity of psicaine in 5 per cent. solution is three-quarters that of cocain hydrochloride; the toxicity of the new alkaloid is therefore identical with that of cocain.

5. It is probable that excitement, fainting, etc., will not be so readily produced by psicaine as by cocain; it may therefore be useful in susceptible persons.
6. For the same reason psicaine should not be used except by those well accustomed to use cocain; the dose of psicaine should not exceed that found safe with cocain.
7. Psicaine is a "dangerous drug."
8. The price of psicaine has not yet been published."

ABSTRACTS

I.—OPERATIONS

(1) **Forster, Andrew E. (Kalmazoo, Mich.)—A review of keratoplastic surgery and some experiments in keratoplasty.** *Amer. Jl. of Ophthal.*, May, 1923.

(1) Forster's paper opens with an interesting survey of keratoplastic surgery dating from 1823 when the first operation of this kind was performed by Reike. The early operations were mostly done with heteroplastic grafts and were almost without exception unsuccessful. Many different methods were adopted. Nussbaum for instance in 1853 used little glass collar buttons 3 mm. in diameter inserted through a trephine hole in the cornea; —infection, iris and lens complications followed. A moderately successful result was obtained by von Haselberg, who in 1913 transplanted a disc of human cornea ½ mm. thick and 5 mm. in diameter on an opaque cornea and secured it with sutures. The graft was opaque at first but clear later, and a year after operation the vision was 1/60, the patient being able to go about alone.

Forster notes that "not only are tissues specific to the species, and so specific that there is no hope of transplanting tissues from one species to another, but it seems now generally agreed that there is an individual specificity within the species itself." A familiar instance of this is afforded by the grouping of cases for blood transfusion. He has, therefore, devised the following technique in conjunction with C. R. Bridgett. An equilateral triangle of 7 or 8 mm. is outlined on the cornea with small-pointed forceps. The entire thickness of the triangle area is cut out, placed in sterile oil and with its angles transposed is again replaced and stitched. The stitches placed at each angle are also sterilized in oil and penetrate all the layers of the cornea and flap. The

operations were performed on cats. The eyes were not irrigated with any antiseptic before or after the operation and the lids were not sewn together as they seemed to remain closed for three or four days after the operation and semi-opened for a week longer. Six animals were operated on and fundus details could subsequently be made out in all cases except one where there was swelling of the lids and conjunctival discharge following the operation.

The idea of the author is that if a macula exists in the centre of the cornea it can be so transposed as to bring a clear portion along the line of vision.

F. A. WILLIAMSON-NOBLE.

(2) **Karelus, Casimar (Cracow).—A new surgical method of treatment in cases of serpent ulcer of the cornea. (Une nouvelle méthode de traitement chirurgical dans les cas d'ulcère serpigineux de la cornée.)** *Rev. Gén. d'Ophtal.*, April, 1924.

(2) After reviewing a small portion of the literature on the subject of serpent ulcer—and an almost exclusively German portion at that—**Karelus** describes his method, which consists in covering the ulcer with two large conjunctival flaps, one from below, the other from above, in such a way that the lower flap slides under the upper and both cover the cornea completely. Before doing this the ulcer is scraped and carefully cleaned up but not, one gathers, cauterized since the object of the operation described seems to be avoidance of the cautery. After the cornea is thus protected Karelus proceeds to excise the lacrymal sac if that organ is the cause of the infection, or to treat the conjunctiva if the infection lies there. Atropin and a double bandage are employed. At the end of a week the sutures loosen and are removed. At the end of ten days the two conjunctival flaps retract and leave, at the site of the ulcer, a strongly vascularized tissue. This vascularization represents the author's object, for the operation was inspired by the fact that an ulcer after travelling across the cornea begins to heal when it reaches the vascular corneal margin. Karelus has performed this operation 24 times for serpent ulcer and gives details of two particularly bad cases.

ERNEST THOMSON.

(3) **Kasass, J. J.—Operation for pterygium. [Zur operativen Behandlung des Flügelfells (Russian.)]** Abs. *Zentralb. f. d. gesam. Ophthal.*, May 20, 1924.

(3) **Kasass** employs for this operation a combination of the methods of Noicewski and Terson. Noicewski does not dissect off

the head of the pterygium with a scalpel, but tears if off with toothed forceps. Terson dissects out a fold of conjunctiva below the pterygium, and fastens it by three stiches to the edge of the conjunctiva above, after the pterygium has been excised. The author tears off the head of the pterygium, and by means of two horizontal and one vertical incision completes the excision of the pterygium. He covers this area by means of a fold of conjunctiva dissected up from below and pulled up over the excised area.

S. SPENCE MEIGHAN.

(4) **Duverger (Strasbourg,) — The shortening of muscles. (Raccourcissement musculaire).** *Arch. d'Ophtal.*, June, 1924.

(4) In 1919* Mettey and Duverger published a description of a method of muscle shortening which they had employed with satisfaction, in cases of strabismus. Since that date **Duverger** has followed the same procedure in all cases of strabismus on which he has operated. He has, however, modified the operation in some details, the most noteworthy of which is the abandonment of silk sutures in the muscle. In their place he uses catgut, which is left *in situ*.

In this communication Duverger describes the operation in detail, and illustrates the method with a number of diagrams. He also gives an account with illustrations of experiments he made on dogs' eyes to show the results of advancement of a rectus muscle; eight months after operation the dogs were killed and sections of the eyeballs made through the insertion of the advanced muscle.

The paper includes brief notes of 39 cases operated upon by the author, and a number of photographs of patients. The results are summarized thus: 39 cases, 29 convergent, 10 divergent.

(*a*) Convergent strabismus: four cases with deviation under 15°, two good results, one moderate, one unsatisfactory; 24 cases with deviation above 15°, 21 good results, three poor results.

(*b*) Divergent strabismus: ten cases, in all of which the immediate results were very good, but in some, from one to five years later, slight divergence re-appeared, though insufficient to mar the aesthetic effect.

Duverger's paper should be read in the original. It is useless to give a description of the operation without the aid of the illustrations; with their assistance the steps of the procedure are easily followed.

J. B. LAWFORD.

*Arch. d'Ophtal., May-June, 1919.

(5) **Lloyd, Ralph I. (Brooklyn, New York).—A factor in infection after needling.** *Arch. of Ophthal.*, September, 1924.

(5) A patient of **Lloyd's** complained of pain in the eye on removing the cover five days after a successful needling. With the loupe a fuzzy end could be seen protruding from the hole made by the needle. On pulling this, it was found to be continuous with the capsular membrane. It was pulled out as far as possible and cut off but the end did not retract into the eye until a swab soaked in pure carbolic was rubbed on it. The string must have been present at the close of the operation, but being transparent it was invisible. The author, therefore, recommends subconjunctival introduction of the needle, or sealing off the limbus puncture with a carbolic swab.

F. A. WILLIAMSON-NOBLE.

(6) **Radcliffe, McCluney (Philadelphia).—Advantages of complete discission by the V-shaped method.** *Amer. Jl. Ophthal.*, October, 1924.

(6) **Radcliffe** favours Ziegler's method of discission on account of its safety, simplicity, freedom from complications and for its preservation of the natural contour of the pupils. The method is as follows: Under cocain anaesthesia and full mydriasis, the cornea is punctured above with a Ziegler knife-needle turned on the flat. The knife is then rotated and the lens punctured with a thrust close to the lower border of the pupil and a little to the left of the middle line. The puncture passes through the entire thickness of the lens. It is then extended as far upwards as possible by a sawing movement in line with the axis of the knife. A similar incision is made starting from a point near the lower margin of the pupil, a little to the right of the mid-line to meet the first, the two forming an inverted V.

Ten cases are reported with good results, the main points being: that only one operation is done, as no subsequent needlings are required; that there is no re-action as the swelling of the lens cortex is in an antero-posterior direction; and, finally, that there is rapid solution of the lens cortex.

F. A. WILLIAMSON-NOBLE.

(7) **Lloyd, Ralph I. (Brookyn, New York).—The Michel metal suture in spastic entropion.** *Arch. of Ophthal.*, November, 1924.

(7) **Lloyd** has found the usual procedure with adhesive plaster unsatisfactory in the treatment of these cases and has resorted to

the use of Michel clips. Two clips are used, "one being placed, about the centre of the lower lid, the other midway to the external canthus." After 48 hours they become loose and are replaced by two others just beside the holes left by removal of the first pair. In cases where a corneal ulcer has developed, as a result of irritation by the lashes of the inverted lid, treatment with clips is required for about a month.

F. A. WILLIAMSON-NOBLE.

(8) **Burlanesco, Cl. (Bucharest).—A novel method of inducing anaesthesia in ophthalmic surgery. (Nouveau procédé d'anaesthésie en chirurgie ophtalmologique).** *Arch. d'Ophtal.*, January, 1925.

(8) Having noted after the use of novocain, certain post-operative disturbances, **Burlanesco** was led to reduce gradually the strength of the injections employed. To his surprise the anaesthesia was as complete as with the stronger solutions.

Following this lead he made a trial of sterile water and obtained excellent results. During eleven months ending October, 1923, he performed 33 ophthalmic operations of various kinds, including excision of eyeball, extirpation of lacrymal sac, removal of dermoid cysts, etc., and 40 removals of foreign bodies from the cornea, without a single failure; the only anaesthetic employed being sterile water.

For deep injections he uses a syringe of 10 c.c. capacity, with a steel or platinum needle 7 cm. in length. Ordinary tap water may be used: it is boiled for fifteen minutes and then allowed to cool to 40° C., so that at the moment of injection it would be about 37° C. The fluid must be injected *very slowly*, "drop by drop," especially during the early stage of injection.

In the case of foreign bodies on the cornea the sterilized water is instilled for five minutes, after which the cornea will be found insensitive.

Burlanesco's explanation of the mechanism of the anaesthesia is that when sterilized water is injected into the deep tissues the anaesthesia is due solely to the compression of the sensory nerve terminals by the fluid. The method being free from all risk of toxic effects seems worthy of trial. Perusal of Burlanesco's paper will supply further details of his procedure.

J. B. LAWFORD.

II.—LACRYMAL APPARATUS

(1) **Barrie, T. Stewart (Glasgow).—Flattening or reduction of the inner canthus following extirpation of the lacrymal sac.** *Glasgow Med. Jl.*, July, 1924.

(1) "The removal of the lacrymal sac does not, as a rule, affect the inner canthus or angle of opening between the lids; in fact there is so little disturbance of the configuration and appearance of the parts surrounding the canthus that one is obliged to inquire into the history of lacrymal cases which come to operation lest an attempt be made to remove a non-existing sac." In the present case **Barrie** describes and illustrates with a photograph the results of a bilateral operation after which, while one canthus presented an undisturbed appearance, the other was flattened out, the commissure being lax but readily drawn into its normal position by slight traction towards the root of the nose. There was also a slight drooping of the margin of the lower lid, similar to that occurring in senile ectropion. The author, after a study of the orbit as described in Whitnall's well-known treatise, comes to the conclusion that the defective result is due to the section going too deep and dividing the pars lacrymalis muscle which ought not to be involved in this operation. "Traced from the lateral side, the muscle fibres of the lids lie in front of the tarsal plates, but at the medial angle of the eye they pass deep behind the sac, or canthus plates, and form the pars lacrymalis muscle, which is attached to the posterior lacrymal crest." Barrie points out, however, that there may be a further explanation in that the orbital septum lies immediately behind the pars lacrymalis muscle, and if this be opened the whole supporting structure will be weakened, and healing will take place with the parts in a faulty position.

ERNEST THOMSON.

(2) **Husson, A. and Jeandelize, P. (St. Quentin and Nancy).—About the lacrymal operation of Dupuy-Dutemps and Bourguet. (A propos de l'opération lacrymale de Dupuy-Dutemps and Bourguet.)** *Rev. Gén. d'Ophtal.*, October, 1924.

(2) This article by **Husson** and **Jeandelize** does not describe in detail the operation of plastic dacryocysto-rhinostomy, but is concerned with a number of details which have emerged in the course of sixty operations of this kind. They modestly say that these sixty cases add nothing to the imposing statistics of the authors of the method, but neverthelss have their value "for they demonstrate that operators who have no operative pretentions can

in the ordinary way and with success perform a plastic dacryocysto-rhinostomy." The authors have paid special attention to the subject of local anaesthesia and what they have to say about this is one of the principal points in this article. They now use three cubic centimetres of allocain to which is added adrenalin. The amount of this latter seems so small that the reviewer prefers to give the French words, viz.: "auxquels nous ajoutons ¼ de milligramme d'adrénaline au millième." Half of the above quantity of anaesthetic is injected above and half below the sac, and along the line of incision may also be injected a cocain-adrenalin solution. The anaesthesia thus produced is sufficient for a long operation. Cocain-adrenalin tampons must also be placed within the corresponding nostril. The remainder of the article is mainly taken up with various difficulties which may occur, such for instance as the presence of sinus cells between the bony wall and the nasal mucous membrane, and with details of some of the cases. Two special cases are related in which relapse occurred shortly after operation. Both of these cases happened to contract coryza. In the one case there had been noticed at the time of operation a sinus cell full of pus, and in the other a *cul-de-sac* had formed which had become re-infected. The first case became completely cured by lavage and injections of argyrol. The second was not completely cured and the authors regard this failure as due to faulty technique on their part and not as in any way reflecting on the reliability of the operation, which, it is obvious, they greatly admire and for the success of which they give all the credit to Dupuy-Dutemps and Bourguet.

ERNEST THOMSON.

(3) **Cange, A. (Athens).—Congenital gangrenous dacryo-cystitis. (Dacryo-cystite congénitale gangréneuse.)** *Arch. d'Ophtal.*, December, 1924.

(3) In the *Annales d'Oculistique*, 1900, Veillon and Morax first described, as a definite clinical entity, a disease of the lacrymal sac to which they applied the term "Peri-cystitis of gangrenous type." **Cange** publishes full clinical and pathological notes of a case occurring in a newly-born child, apparently the first recorded example in infancy. His patient came under observation when one month old, the disease, according to the mother's statement having appeared fifteen days previously. In spite of active treatment by surgical measures and by serotherapy a fatal result ensued nine days after the child was first seen. There is no note of a post-mortem examination. The clinical picture was that of a large crater-like area in the region of the sac, with much surrounding oedema; the floor of the crater covered by a greyish slough with offensive odour.

Bacteriological examination of the exudate and tissues showed: (1) Large numbers of a feebly-staining gram-negative spirillum, with 3-4 spirals, morphologically resembling the spirillum of Vincent; (2) very numerous fine bacilli, 2-3 μ, gram-negative, immobile and without visible spores; (3) numerous cocci, gram-positive, of the staphylococcus type.

No growth of the spirillum could be obtained on any culture medium, aerobic or anaerobic. The bacilli grew slender colonies, greyish and whitish, but only in anaerobic cultures. From the cocci abundant cultures were obtained, aerobic and anaerobic, liquefying gelatine, and resembling cultures of staphylococcus aureus. Intravenous injection of the cultures was fatal to rabbits in four days.

J. B. LAWFORD.

(4) **Bollack, J.—X-ray examination of the lacrymal passages by injection of iodized oil. (Sur l'exploration radiographique des voies lacrymales par l'injection d'huile iodée.)** *Ann. d'Ocul.* Vol. CLXI, p. 321, 1924.

(4) In the examination of the lacrymal passages by means of the X-ray, the substance for injection requires the following characteristics:

(*a*) It must be opaque to the rays, even in small quantity;
(*b*) it must be perfectly homogeneous;
(*c*) it must be fluid at the temperature of the body; and
(*d*) it must be non-irritating and non-painful.

Bollack claims that this lipoidol, made by Lafay, containing 40 per cent. of iodine, is a suitable oil. After cocainization of the conjunctiva, in a case of lacrymal obstruction, the sac is expressed, then irrigated with normal saline, expressed again, and the fluid lipoidol injected, after having been slightly warmed, by means of an ordinary syringe. Usually about 0.5 c.c. is sufficient.

Bollack claims for the method both that it is a simple method of investigating the normal lacrymal passages, and also that, in disease of the passages, it affords exact indications of the changes of calibre, distension or contraction, the extent and position of the obstruction, and the condition of the neighbouring parts.

HUMPHREY NEAME.

(5) **Bruner, Abram B. (Ohio).—Tumours of the lacrymal gland.** *Amer. Jl. of Ophthal.*, October, 1924.

(5) **Bruner** after noting that there is a death rate of 12 per cent. in cases of lacrymal tumours, states that the majority of new growths have been described as endotheliomata, sarcomata, carcinomata or mixed tumours. He describes two cases.

Case 1 was a man, aged 22 years, with a history of increasing swelling of the left upper lid for the previous three weeks; there was considerable increase in size of the growth during a further period of three weeks, and it was removed through an incision in the upper lid. Macroscopically it was soft, lobulated, and involved the whole gland. Sections showed the tumour to be composed of numerous new-formed connective tissue cells, containing fat, there was no definite capsule, and it was classified as a fibro-myxo-lipoma.

Case 2 was a youth aged 17 years. Swelling had been noted for a year previously. The eye was displaced forwards, downwards and inwards, and there was moderate ptosis. A firm nodular mass could be felt in the upper fornix after cutting through the external canthus. The tumour contained spindle cells with oval nuclei, masses of cartilage, bony trabeculae, gland-like structures lined by various types of epithelium, and epithelial pearls. There was a definite capsule in no place invaded by tumour tissue. The diagnosis was teratoma.

F. A. WILLIAMSON-NOBLE.

III.—OCULAR SYPHILIS

(1) **Blatt N. (Roumania).—Clinical observations on late syphilitic affections of the fundus.** (Eye Hospital, Zenica-Bosnia.) **Klinische Erfahrungen über spätluetische Augenhintergrundserkrankungen in Bosnien.**) *Arch. f. Ophthal.*, Vol. CIV, Pt. 3, 1921.

(1) **Blatt** had the opportunity of examining 200 patients in Bosnia, among whom he noted twelve cases of retino-choroidal disease, whereas optic nerve atrophy from tabes or paralysis among Bosnians or Turks was observed neither by him nor by his predecessor, Professor Salus. Nor were other eye-symptoms noted which could be accounted for by tabes or paralysis. The chorio-retinal changes did not appear as complications in the secondary period, as in middle Europe, but as a late syphilitic development. Apart from the unusual time at which the complications appeared, the cases were characterized by advanced vascular changes and marked connective tissue formation in the fundus. Wassermann's reaction was positive in all cases but one. Corneal disease was present only in one instance, and assumed the form of a right-sided parenchymatous keratitis, which healed rapidly under anti-syphilitic treatment. Remarkably frequent was inflammation of the ciliary body, also of the iris, and punctiform

opacities of the vitreous body were invariably present. Actual disease of the papilla was never observed. The retino-choroidal foci were partly pigmented, and more numerous at the periphery than at the posterior pole: the choroidal vessels were often completely sclerosed. The new-formed connective tissue between retina and choroid had a remarkably shiny appearance, and was frequently to be seen also on the surface of the retina. Blatt proposes for the cases he has described (which are also in part illustrated) the name of "Retinochoroiditis fibrosa luetica."

V. ST. JOHN.

(2) **Blatt, N. (Roumania).—Examination of the liquor cerebro-spinalis as a diagnostic factor in syphilitic affections of the eye. (Uber die diagnostische Verwertung der cerebro-spinalis Liquorbefunde bei luetischen Augenerkrankungen.)** *Arch. f. Ophthal.*, Vol. CVI, 1921.

(2) **Blatt** examined the liquor cerebri in seventeen cases of syphilitic affections of the eyes, and lays stress on the diagnostic value of this examination. The result was negative in cases of disease limited to the eye (parenchymatous keratitis, iritis, choroiditis) as well as in several cases of optic nerve atrophy; in other optic nerve cases and in reflex immobility of the pupil, it was positive. He then makes a summary of the different ocular affections in which the liquor has been examined.

V. ST. JOHN.

(3) **Kafka, V.—The physiology and pathology of the liquor cerebro-spinalis with special reference to ophthalmology.** *Zentral. f. d. Gesamte Ophthal.*, Vol. X, June, 1923.

(3) **Kafka** considers that the diagnosis and prognosis of ocular affections by means of the liquor cerebro-spinalis as demonstrated by Blatt's publication ("On the examination of the Liquor Cerebro-spinalis as a Diagnostic Element in Syphilitic Affections of the Eye" noticed above) is of great value. He comes to the following conclusions:

1. Liquor cerebri giving a positive Wassermann can not only settle the diagnosis of a neuro-syphilitic affection of the eye, but can also offer indications which are very useful in localizing the central or nervous lesion responsible for the ocular changes.
2. A positive liquor is always of value, a negative liquor only when the blood gives a positive Wassermann.
3. A positive liquor unquestionably indicates syphilitic disease of the central nervous system, but the contrary does not necessarily hold good in case of a negative liquor.
4. A positive liquor does not indicate, in the absence of other signs, that we can expect disease of the central nervous system to

develop in the course of time. It points rather to already existing syphilis of the central nervous system which, so far, has not betrayed its presence by other clinical symptoms.

V. ST. JOHN.

(4) **Blatt, N. (Roumania).—The practical value of the Sachs-Georgi reaction in syphilitic diseases of the eye. (Die praktische Verwertbarkeit der Sachs-Georgischen Reaktion bei den luetischen Augenerkrankungen.)** *Klin. Monatsbl. f. Augenheilk.*, Vol. LXIX, 1922.

(4) The comparative value of the Wassermann and Sachs-Georgi reactions was tested in 70 cases (50 syphilitic and 20 non-syphilitic). Of the 20 control cases 18 showed a negative result, in the remaining two the reaction was doubtful. In ten cases of parenchymatous keratitis there was present in six cases congenital syphilis, and, what is somewhat striking, in four cases the disease was acquired. The cases of congenital syphilis gave, with a single exception, positive results with both tests, while in the cases of acquired syphilis more negative than positive results were obtained. One case gave a negative Wassermann and a positive Sachs-Georgi Reaction. Also in the other categories the two reactions mostly gave results which agreed. A striking superiority of one reaction over the other was not noted. A slight superiority in favour of the Sachs-Georgi was observed in affections of the uveal tract, as 33, 3 per cent. positive Wassermanns could be opposed to 38, 8 per cent. positive Sachs-Georgi Reactions. In cases of disease of the retina and optic nerve, on the other hand, the author noted 58, 3 per cent. positive Wassermanns and only 5 per cent. positive Sachs-Georgi Reactions, while in affections of the pupillo-motor tract both reactions were positive in 60 per cent.

V. ST. JOHN.

(5) **Ling, W. P.—Primary chancre of the palpebral conjunctiva.** *Jl. of the Amer. Med. Assoc.*, August 16, 1924.

(5) Ling of Pekin reports a case in which there was a double infection of the tarsal conjunctiva and the skin of the forehead. The possible specificity of lesions of the lid and conjunctiva should be borne in mind when a diagnosis is being made. The author quotes Maxey (*Amer. Jl. of Ophthal.*, 1918), who compiled the literature of 82 cases of primary syphilitic lesions of the eye and its appendages. *The Bull. of the Ophthal. Soc. of Egypt*, 1923, contains an important paper by Sadek, who reports 57 cases which occurred in his own hospital practice, and quotes the *Amer. Encyc. of Ophthal.*, Vol. III, tabulating 372 cases.

A. F. MACCALLAN.

(6) **Cosmettatos, G. F. (Athens).—Affections of the cornea in acquired syphilis. (Des affections de la cornée au cours de la syphilis acquise.)** *Arch. d'Ophtal.*, December, 1924.

(6) Primary chancre of the cornea is extremely rare. Only one example, published by Binet in 1883, is known to **Cosmettatos** and there is some reason to doubt the primary nature of the lesion in this case. In the secondary and tertiary stages the cornea is not infrequently involved, the disease appearing as diffuse parenchymatous keratitis, punctiform keratitis, or gumma, the first-named being the most common.

Cosmettatos publishes notes of four cases of the punctiform variety occurring in two males, aged 23 and 30 years, and two females, aged 29 and 40 years. The affection was bilateral in one patient, unilateral in the other three. In two the transparency of the cornea was completely restored; in the other two partial recovery ensued.

Two examples of gumma of the cornea are also recorded by the writer. Both patients were women, aged 35 and 40 years, and in both vigorous anti-syphilitic treatment was followed by complete recovery.

J. B. LAWFORD.

(7) **Margerin, R.—(Clinique du Dr. Chaillous aux Quinze-Vingts.) On the treatment of interstitial keratitis with bismuth salts. (Du traitement des Kératites Interstitielles par les sels de Bismuth.)** *Ann. d'Ocul.*, Vol. CLXI, p. 355, p. 1924.

(7) **Margerin** used iodo-bismuthate of quinine in oily suspension called "Quinby," prepared by Aubry, in ampoules of 10 centigrams of the salt per c.c. (=2.5 c.gm. of bismuth metal). Only very occasional complications arose as the result of use of this preparation, in the form of swelling of the gums in children with defective teeth, but no marked stomatitis. No urinary changes developed, but, rarely in children, some fatigue without any obvious wasting.

Dosage. The dose for adults is 20 to 30 centigrams of bismuth per week up to a total of 2 to 3 grams; for children 8 to 17 centigrams per week. The drug is administered in one or two doses per week, for not more than six to eight weeks, for fear of development of bismuth anaemia. Often in addition to the above, doses of 15 centigrams of neosalvarsan (914), to stimulate blood formation in contrast to the effect of the bismuth, are given.

Margerin reports six cases of established bilateral interstitial keratitis in which he administered this treatment, and in which there was no definite improvement under other accustomed methods of treatment. In these established cases, there was no marked change of progress by treatment with the bismuth.

In eight cases of early unilateral interstitial keratitis, however, the following were noted:

(1) Marked diminution of photophobia and pain within about three weeks.

(2) Curtailment of the duration of the disease.

(3) Absence of disease development in the second eye, except in two cases out of the eight.

It should be noted that of the six cases in which one eye only was involved, the age of the patient was six, seven, seventeen, eighteen, twenty and twenty-seven years respectively, and that these cases were under observation for an average of only 2.1 months.

The two in which the second eye became involved, as is stated, to a much less extent than the first eye, were aged seven and eight years respectively, and suffered from the appearance of the disease in two and a half and four and a quarter months respectively from the commencement of treatment. These two cases were under observation in all up to three and a quarter and five months respectively.

HUMPHREY NEAME.

BOOK NOTICE

Bulletin of the Ophthalmological Society of Egypt, 1924.

This volume, containing the communications read at the annual meeting of the Ophthalmological Society of Egypt, on March 7, 1924, has just been issued. Besides the delay in publication, we must criticize the proof-correction. As the result of remarks made in these columns with regard to the Bulletin of 1919, there was a great improvement in the volumes from 1920 to 1923, which, however, has disappeared in the present volume. We may remind the Editorial Committee of the Bulletin that it is not customary to underline italicized words and sentences, nor when a list of abbreviations is appended, to include in the text additional ones. By a comparison with the report of the first meeting of the Society in 1903 we note that only two of the eight existing original members of the Society are asterisked as such. The Bulletin contains some interesting papers, which are very creditably written, considering that they were not in the mother tongues of the authors.

E. Fuchs, in a paper on "Chronic Primary Glaucoma," describes the case of a middle-aged lady, who for some years was suffering from frequent sick headaches according to some of her doctors, from neuralgia of the fifth nerve according to others. She was repeatedly examined by very experienced eye specialists, who

never found anything wrong with the eyes. Acuity and field of vision as well as the fundus were normal, tension was mostly between 12 and 15 mm. Hg. The patient had never noticed coloured haloes, and still it was a case of glaucoma and an iridectomy in both eyes stopped at once all the complaints of the patient, who is in perfect health twelve years after the operation. The original failure in diagnosis was due to the fact that the patient had never been examined during one of the attacks of pain. It illustrates also the fact that in chronic glaucoma the tension in the intervals between the attacks may be very low. It is also remarkable that in this case there was never blurred or coloured vision during the attacks but only pain, and that the patient did not develop glaucomatous cupping in spite of the long duration of the disease.

The author states that a case of primary glaucoma is not rarely mistaken for atrophy of the optic nerve if the optic disc is only pale, not cupped. He has recorded quite a number of such glaucoma cases, in which the excavation developed only when the eye was almost or completely blind, or in which the true nature of the disease revealed itself by a final attack of acute glaucoma. He examined several of these cases microscopically and found that the lamina had resisted the increased pressure while the nerve fibres of the optic nerve head had disappeared. An early diagnosis of such cases can be made only by repeated tonometric observations. von Graefe pointed out more than sixty years ago that after reduction of the tension by an operation the glaucomatous cupping may disappear, but he was also aware that it might reform later on. von Graefe accounted for the disappearance of the excavation by supposing that after relieving the tension the lamina cribrosa was allowed to go back to its normal place. Fuchs had the opportunity of examining such cases microscopically, and can state that the lamina cribrosa still retained its excavated position, but that the nerve head in front of the lamina was highly oedematous and swollen, so as to fill the cup. Fuchs considers this oedema as being due to lymph-stasis in the papilla. Within the optic nerve the lymph current is believed to proceed intracranially and empty into the cerebro-spinal fluid. The centripetal direction of the lymph current is due to the fact that the intraocular tension is higher than the intracranial pressure. Lymph-stasis must occur when the difference in pressure is abolished or reversed, when the intracranial pressure has risen or the intraocular tension has dropped. Hence one may see in cases of reduced intraocular tension, as for instance subsequent to fistulization of the cornea, the picture of choked disc, if ophthalmoscopic examination is possible. The same may take place when the tension is low after an operation, the papilloedema manifesting

itself not as a prominence of the disc, but as a disappearance of the glaucomatous excavation. When later on, after cicatrization of the wound, the tension goes up to normal, the glaucomatous excavation re-appears.

Fuchs describes the non-operative methods of reducing the intraocular tension. Among these he has used two methods for reducing the tension in cases of acute glaucoma previous to operation. One is venesection by which he has reduced the tension from 50 mm. Hg. to 25 mm. Hg. The other is by the intravenous injection of 200 to 250 c.c. of a 10 per cent. solution of sodium chloride, an effect which can be attained by getting the patient to swallow 20 to 30 grammes of the same salt in solution.

Glaucoma and High Myopia. Fuchs states that owing to the thinning of the sclera at the posterior pole of the eye in high myopia, a glaucomatous excavation may extend beyond the temporal margin of the papilla over to the adjoining part of the sclera, so that the excavation is composed of two portions, the nasal one corresponding to the optic disc, the temporal one to the adjacent sclera. The limits between these two portions are marked only by the difference of the slightly pink disc on the one hand and the white sclera on the other. The temporal margin of the excavation appears as a crescent-like ridge dividing the staphyloma into a nasal (cupped) and a temporal (uncupped portion). According to his experience only a few of such cases are diagnosed correctly as simple glaucoma by means of the tonometer and by the condition of the field of vision.

Meyerhof has found that the coincidence of "Glaucoma and high Myopia" is not infrequent in Egypt. He has seen twenty-six cases of which he has exact observations. Only two of these were under 40 years of age. The onset was always slow and lingering without headaches or haloes. All these patients whose vision was already defective complained of a slow diminution of vision and nothing else. The field of vision does not always show any characteristic sign; Bjerrum's para-central scotoma may be caused by the peri-papillary and central stretching of the choroid. Cupping of the disc was only found in six of the twenty-four cases. The prognosis of these cases, under treatment but without operation, is better than that of ordinary primary chronic glaucoma.

In the discussion on "Rasheed's cases of Extraction of Immature Cataract," Fuchs reminded the Society that the condition of the posterior cortex only was of importance. If this is opaque, the cataract may be operated on safely, even if all the rest of the lens is transparent. The operation for ripening a cataract bears only on the anterior cortical substance, which is broken up by the massage of the lens and becomes opaque, while the posterior cortical substance which alone matters remains unaffected.

"Operations for Entropion and for Trichiasis" were described by Sander and by Khairat. The general opinion of the Society appeared to be that they were inferior to the well known and satisfactory operations of Snellen (removal of a wedge-shaped piece from the anterior surface of the tarsus, together with the well recognized modifications), of van Millingen (insertion of a mucous membrane graft into a groove along the edge of the eyelid), and the tarsectomy of Heisrath.

A case of "Leprosy of the Cornea" was reported by Meyerhof and Sobhy; the latter also makes some observations on "Spring Catarrh in Egypt."

"Trachoma in Finland" is the title of a paper by Enroth. This author states that 4.5 per cent. of the population of 3½ millions are infected with this disease. Infection takes place apparently at a later period in life than in Egypt, as below five years of age there is only an incidence of 1 per cent.

"A Case of Primary Tumour of the Optic Nerve" is described macroscopically by Kattan, a report which it would have been preferable to have deferred until complete microscopical examination had been effected. "Three Cases of Papilloedema in which the Skull was Trephined" are described by Bakly. In one of these good vision was retained.

A. Barrada relates details of a number of interesting functional conditions.

NOTES

Death

The death of the RIGHT HON. SIR CLIFFORD ALLBUTT, K.C.B., P.C., should not pass unnoticed in an ophthalmic journal. A pioneer in advocating the importance of ophthalmoscopic examination in cases of general disease; his work on "The Use of the Ophthalmoscope in Diseases of the Nervous System and Kidneys" appeared in 1871: nor is it usually remembered by the present day members of the medical profession that to him is due the small clinical thermometer in daily use. He succeeded Sir George Paget in 1892 as Regius Professor of Physic in the University of Cambridge after having built up a great reputation as a consulting physician in Leeds.

The last thirty years of his life, spent in the university atmosphere and surrounded by young men, kept him young himself. Although not quite a Peter Pan who never grew up, Sir Clifford Allbutt certainly did not appear to grow old. He was in his 89th year and died suddenly in his sleep.

Appointment

MR. F. A. WILLIAMSON-NOBLE has been appointed Representative of the Ophthalmic Section on the Library Committee of the Royal Society of Medicine; and honorary librarian of the Bowman Library.

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Ophthalmology in Mexico

WE have received a copy of the first number of a new journal in ophthalmology published in Mexico under the title *Revista Oftalmologica del Hospital de Ntra Sra de La Luz.*

This is a tri-monthly publication edited by Drs. Enrique Graue; Rafael Silva; Louis S. Viramontes and Daniel M. Vélez. The price is not stated. Part I forms a brochure of 20 pages, and deals mainly with ophthalmia neonatorum. There is also an editorial and some introductory remarks by Dr. Daniel M. Vélez.

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Ophthalmological Research

A FUND has been raised, consisting of $3,000,000 for the establishment at Johns Hopkins University of a centre for ophthalmological research to be called the Wilmer Institute.

The General Education Board gave half the money and the rest was subscribed by friends and patients of Dr. William Holland Wilmer, of Washington, who will retire from private practice in order to undertake the Directorship of the new centre.

The Wilmer Institute will consist of laboratories and of a hospital with 60 beds, 40 of which will be reserved for non-paying patients. It is hoped that the new institute will be ready by September.

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Greek Epigram on an Ophthalmic Surgeon.

WE are indebted to Mr. Percival for the following delightful epigram by Nicarchas on Simon the Greek ophthalmic surgeon.

Ἤν τιν' ἔχῃς ἐχθρόν, Διονύσιε, μὴ καταράσῃ
τὴν Ἶσιν τούτῳ μηδὲ τὸν Ἁρποκράτην
μηδ' ἔι τις τυφλοὺς ποιεῖ θεός, ἀλλὰ Σίμωνα
καὶ γνώσῃ τί θεὸς καὶ τί Σίμων δύναται.

"If you have any enemy, Dionysius, call not down on him either Isis, Harpokrates or any other god who strikes men blind, but Simon, and you will know what god and what Simon can do."

Royal London Ophthalmic Hospital Dinner

THE Annual Dinner of the Royal London Ophthalmic Hospital was held at the Langham Hotel on Thursday, February 12. The Chairman was Mr. W. T. Holmes Spicer, Consulting Surgeon to the Hospital, and there were present a good number of past and present students.

Among the guests were Lady Cooper, Lady Lister, Sir Humphry Rolleston, President of the Royal College of Physicians, Vice-Admiral Joseph Chambers, Director-General of the Medical Department of the Navy, Lieut.-General Sir W. B. Leishman, Director-General, Army Medical Service, Air Commodore D. Munro, Director of the Royal Air Force Medical Service, A. E. Hayward Pinch, Esq. J. S. Fraser, Esq., Chairman of the House Committee of the Hospital, and Professor Elliot Smith.

The speakers were the Chairman, Sir Humphry Rolleston, Sir W. B. Leishman, Mr. Fraser, Mr. Hepburn, Sir John Parsons, and Mr. Fisher. Mr. O. Gayer Morgan sang some songs.

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North of England Ophthalmological Society

THE following officers were elected to the Council at the meeting of the North of England Ophthalmological Society on March 6, 1925: President: Mr. Harry Lee (Leeds); Vice-president: Mr. Edgar Stevenson (Liverpool); Treasurer: Mr. S. D. Lodge (Leeds); Secretary: Mr. Percival J. Hay (Sheffield); Members of Council: Mr. H. Holmes (Liverpool), Mr. W. Barrie Brownlie (Blackburn).

FUTURE ARRANGEMENTS

1925.

April 7.—Midland Ophthalmological Society, at Leicester.
May 8.—Section of Ophthalmology, Royal Society of Medicine.
May 9-14.—Congrès de la Société française d'Ophtalmologie, at Brussels.
June 2.—Midland Ophthalmological Society, at Oxford.
June 12.—Section of Ophthalmology, Royal Society of Medicine (Annual).
July 14-17.—Convention of English-speaking Ophthalmological Societies, in London.
July 21-24.—British Medical Association, at Bath.
October 6.—Midland Ophthalmological Society (Annual), at Birmingham.